Report on UC-ARB Conference

"TECHNICAL BASES FOR CONTROL STRATEGIES OF PHOTOCHEMICAL OXIDANT: CURRENT STATUS AND PRIORITIES IN RESEARCH"

December 16-17, 1974

UC Riverside

California Air Resources Board

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Statewide Coordination for Air Pollution Research

STATEWIDE AIR POLLUTION RESEARCH CENTER

UNIVERSITY OF CALIFORNIA, RIVERSIDE

James N. Pitts, Jr.

Director

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INTRODUCTION AND ACKNOWLEDGMENTS

During 1974-1975, the California Air Resources Board, under ARB Contract No. 3-974, cosponsored with the University of California, through the Statewide Air Pollution Research Center (SAPRC) and Statewide Coordination for Air Pollution Research, a series of "state-of-the-art" conferences dealing with current priorities in air pollution research and control. These conferences stressed technical discussions and focused on new research needed to develop improved control strategies.

Details of the conferences are summarized below.

Date, Place, and Chairman	Title
March 18-19, 1974 Berkeley, California James N. Pitts, Jr., Chairman	"Chemical and Physical Interactions of SO_x, NO_x, and Photochemical Oxidant: Current Status and Priorities in Research"
May 20-21, 1974 Davis, California O. Clifton Taylor, Chairman	"Photochemical Smog--Effects and Causes in Agriculture: Current Status and Priorities in Research"
December 16-17, 1974 Riverside, California James N. Pitts, Jr., Chairman	"Technical Bases for Control Strategies of Photochemical Oxidant: Current Status and Priorities in Research"
March 25, 1975 Irvine, California T. Timothy Crocker, Chairman	"Health Surveillance Related to Air Pollution"

For informational purposes, the programs of each of these conferences and the lists of participants are included with this report as Appendix A. The abstracts of the Berkeley and Davis Conferences are included as Appendix B. Programs, speakers' abstracts and attendee lists were made available as part of the participant's package received at each of the UC-ARB Conferences listed above. Reports of the conferences appeared in the Statewide Coordination Newsletter published by the Statewide Air Pollution Research Center for California recipients of the CALIFORNIA AIR ENVIRONMENT in the April 1974 and May

1975 issues. Copies of the pertinent portions of the Newsletter are included as Appendix C.

The main body of this report contains the material presented by the speakers at the December 16-17, 1974 Conference, Riverside, California, "Technical Bases for Control Strategies of Photochemical Oxidant: Current Status and Priorities in Research." Transcriptions of the speakers' presentations were submitted to the speakers; their resultant revisions comprise the material in the report of the December 1974 meeting and therefore contain data current at that time.

We gratefully acknowledge the contributions to this series of UC-ARB Conferences by the following people and organizations:

- The California Air Resources Board for their financial support and participation in the programs. We particularly thank Dr. Jack Suder, Project Monitor, Research Section of the ARB.
- Dr. William C. Kuby, Associate Professor, Mechanical Engineering, University of California, Santa Barbara, and then Associate Director of Statewide Coordination for Air Pollution Research, for his guidance in planning and conducting the conferences in liaison with Dr. Suder.
- Dr. O. Clifton Taylor and Dr. T. Timothy Crocker for serving as Chairmen of the conferences on the Davis and Irvine campuses, respectively.
- The speakers and discussion leaders for each of these conferences who gave their time and effort in preparation of presentations which stimulated meaningful discussions of problem-oriented research directed toward the control of air pollution.
- The speakers of the December 16-17, 1974 Conference for their cooperation in reviewing transcriptions of their presentations and submission

of their materials which form the main body of this report. We particularly thank and gratefully acknowledge our indebtedness to A. P. Altshuller, David V. Bates, Donald L. Blumenthal, Basil Dimitriades, George J. Doyle, Alan Eschenroeder, John R. Kinosian, John H. Knelson, Robert G. Lunche, Robert E. Neligan, Henry K. Newhall, Robert F. Sawyer, and Ralph C. Sklarew.

- The personnel of SAPRC's Office of Technical Information and Statewide Coordination for Air Pollution Research for preparation of materials and making arrangements for the conferences and this final report.

James N. Pitts, Jr.
Director
Statewide Air Pollution Research Center
and
Statewide Coordination for
Air Pollution Research

HEALTH EFFECT IMPLICATIONS OF AMBIENT AIR CONCENTRATIONS OF OZONE ALONE AND IN COMBINATION WITH SULFUR DIOXIDE

by

David V. Bates, M.D.
Dean, Faculty of Medicine
The University of British Columbia
Vancouver, B.C., Canada

Scientists interested in ozone at this point of time seem to be divided into two rather distinct groups - there are those concerned that it may be diminished in the stratosphere due to man's action, and hence its essentially protective effect may be reduced; and there are those concerned that it may increase at ground level due to dangerous concentrations of some pollutants. They are, therefore, looking at the problem in terms of too much ozone. I am going to deal only with the second group of people - (those concerned that we may have too much of this gas at ground level).

My interest in ozone actually did begin in relation to the first of those topics and not the second. Some early work from the German V2 rocket program became available to us in London in the late 1940's, and showed for the first time the stratospheric ozone concentration. At a height of 30 kilometers down to 15 kilometers, there were high concentrations of ozone. My interest was precipitated by a request to consider some of the physiological implications of the Comet 1 aircraft. One of the questions that came up was whether this aircraft would compress into its cabin a sufficient quantity of ozone to be a hazard. By the time I moved to Canada in 1955, this was beginning to be an interesting question. I didn't get around to doing anything about it until 1960 when the first DC-8 was

delivered to Air Canada. With the help of the Chief Engineer of Air Canada whom I knew, I set up an experiment putting rubber bands in either a sealed container or an open container in three different locations of the DC-8, under the navigator's desk, in the passenger compartment, and right in the rear in one of the toilets. We also put the same container in the nose wheel compartment so that it was exposed to the ambient atmosphere. We found that we could easily detect early cracking of the rubber in the open, as opposed to the closed, container on the flight deck. It took us about six months to get an approximate idea of what the concentration of ozone must have been for the hours the aircraft had spent above 30,000 feet. It was two years later that the FAA, using much better equipment and doing a much more thorough experiment, confirmed that the ozone level was somewhere about .15 to .20 ppm. I mention this because, as far as I am aware, there still has been no study to see whether these concentrations on the flight deck of these aircraft are having any long-term effect on the air crew.

Our next study was concerned primarily with people who were working in a high ozone environment. We studied welders who were using high intensity arc welding doing pulmonary function studies on them looking for any effects of long-term hazards. We could find none.

The first point I want to make is that what appeared to be a localized problem ten years ago, is rapidly becoming a general one. Figure 1 is a photograph taken last January of the City of Calgary, when there was snow on the ground, showing the very clear inversion layer in which the automobile emissions are trapped. Although there can be levels of NO_2 in this atmosphere, there isn't sufficient sunlight intensity at this time of year in Calgary to take the reaction forward into the photochemical series of

reactions which would otherwise occur. Figure 2 shows a natural gas burning electric generating plant very unfortunately located in respect to the city of Edmonton, and this has, of course, a very formidable output of oxides of nitrogen. In fact, for the City of Edmonton 46% of oxide of nitrogen emitted in the whole city is from power plants. And as a matter of fact, 46% is from that particular power plant, as far as I can tell, motor vehicles accounting for only 31%. So that where you are burning very large amounts of natural gas, you will have a point source of oxide of nitrogen emission. In Edmonton, in January, the sunlight intensity is not enough to take this reaction forward. It is for four months in the summer, but it is not at this time in the winter. In the winter of 1970-71 in Edmonton they had a series of inversions, and the mean monthly oxides of nitrogen level went up to 0.15 ppm, which is high even in Chattanooga terms. So that we have particular problems in Canada mostly related to the fact that we have a lot of natural gas. We do know in Vancouver that we can get the same peaks of oxides of nitrogen in Vancouver, though the data is sparse and incomplete. Problems of ozone pollution are not limited to affluent countries. A physician working with me on ozone who came originally from Teheran brought back two very nice photographs taken from the balcony of his parents' apartment in Teheran. In the first one, taken at 7:00 in the morning as the sun was just coming up, with the hills around Teheran very nicely shown, the scene looked very much like California. In the second one, the view from the same balcony at lunch time showed the hills completely obscured. He was sufficiently aware of photochemical pollution to be sure this was the problem. Obviously the problem is a general one.

A paper in Nature last year by Derwent and Stewart showed for the first time recordings of nitric oxide, nitrogen dioxide, and ozone, right in the center of the City of London, for three days in July of 1972. Sulfur dioxide was present as well. So that even the old cities that have had a "reducing" pollution problem with SO_2 and acidity, are now moving into an era in which the sequence of gases first described in Los Angeles is apparent over a three-day period. One of the reasons we are now getting this data, of course, is that we can now measure ozone reliably in the presence of sulfur dioxide whereas before this was difficult. So much for the generality of the problem. The point that I take from it is that we have to be very much concerned with mixtures of pollutants.

Let me turn now to acute effects, in some ways the easiest to talk about. Figure 3 shows the effects on one test of lung function of 0.37 ppm of O_3 and 0.37 ppm of SO_2 when present together. There is a very much greater effect when both gases are present than was observed when only a single gas was present. This work needs extension and repetition; yet it is sufficiently striking to suggest that we have a new phenomenon to deal with when both gases are present together. We are becoming very aware that the tests we have used up to this point have concentrated primarily on the large airway, and that the small airways could be affected adversely without the tests of function which have been popular, like the single expiration test, measuring it. We will clearly have to employ all types of test, and particularly those that measure changes in small airways to be sure of the dose of a gas that is without an acute effect.

We have recently been doing a collaborative experiment with Dr. Hackney of Rancho Los Amigos which is beginning to show results of very considerable interest. Dr. Hackney is scheduled to read a paper on our preliminary results at the May meeting of the American Lung Association in Montreal; at this point in time it seems fair to say this - that we have established that the same Canadians tested in Canada or in Los Angeles have the same response to ozone; our Mast ozone meters in Canada are within 10% of the chemiluminescent meter in the chamber at Los Angeles; and the change in pulmonary function seems greater in the Canadians than that seen in normal subjects selected from the population living in Long Beach. This statement seems to be true also of hematological changes consequent upon O_3 exposure. Now this is, of course, very challenging information. It is too early to conclude that the results could only be explained by the development of tolerance. Is it "good news or bad news"? The answer has to be "we don't know." Because although the diminished response could be called tolerance on the one hand, we don't know whether there is any long-term biochemical price you may be paying for that tolerance. So that, at this point in time, we should not try to state whether it is good news or bad news, but we should try to make sure that it is really "news." There is still a considerable amount of collaborative work between these different laboratories to be done to clarify exactly what is happening. A second issue, and an extremely important one, has to do not with assessing the importance of these acute effects. I may say, in passing, that the laboratory acute effects are perfectly replicated in the literature which describes people, particularly children, in acute episodes of oxidizing air pollution. The short cough, the pain under the sternum, the obvious difficulty there is in kind of describing exactly why it is so unpleasant,

all of these come clearly through the more perceptive reports, particularly from Japan. Those who believe them to be exaggerated should exercise in ozone in a controlled environment when they will find to their surprise, as I have found, that the pain, for example, which occurs with the acute irritation of the trachea, is not related to breathing. It is independent of the phase of breathing and it is moderately severe, being more than just a discomfort. If you have experienced it you will understand why someone who has had a coronary may think that he is having another one. Similarly, the cough is very hard to control and is precipitated by taking a deep breath and exaggerated, therefore, by exercise. This again is a distressing symptom in those who breathe enough ozone to bring it on. There are persistent reports of malaise following ozone exposure, they go back a long way but we are a long way from understanding why there should be malaise. All the early literature of ozone exposure describes a syndrome very like influenza ending up with a headache and nausea and so on (these are probably very high exposures) but we don't understand the genesis of those effects at this point of time. Nor are we keen to replicate them because the concentrations you would need to use would, I think, be dangerous. The third main question has to do with the important problem of aging. If you measure the transpulmonary pressure in the human lung related to lung volume, you will find that as we go from mean age 20 to mean age 70 that the human lung undergoes a fairly steady change in this relationship so that the 70-year-old lung has a higher residual volume (that is, lung volume at 0 pressure). Also the shape of the curve is changed, so that at lower pressure he is closer to 100% of lung volume than somebody younger. This has been known for sometime, but we have only

recently become aware of its general significance. We now understand that that aging effect of a loss of elasticity, a loss of recoil, lies behind a good many of the phenomena that distinguish a 70-year-old from a 30-year-old and accounts for the steady lowering of arterial oxygen tension that occurs with age.

In terms of ozone, the work by Donald Bartlett at Dartmouth has opened this whole question anew. After exposing young rats to 0.3 ppm for a 30-day exposure, he found that there was displacement of the pressure/volume curve of the lung. He was very careful to point out, however, that he has observed displacement of the top point, the level of lung inflation at maximal negative pressure to be highly significant but there is not a total displacement of the curve which you would have expected if the aging effect was being precisely duplicated. He was able to show that this was without any morphological correlate, that the alveoli of the lung had grown normally and were normal in number and in size. So this is a phenomenon in which the lung reaches a higher volume at a given pressure in the exposed animals compared to the controls. I have spent a little time on this because there are reasons to suggest, armchair reasons, that ozone may be particularly good at affecting the elasticity of an organ like the lung. Its particular effect on rubber, that I began showing you, is by unhooking the linkages on which the elasticity of natural rubber depends. It is easy to make these analogies but obviously, from what I have shown you, we have a long way to go yet before we are in a position to understand the answer as to what the long-term effect of ozone might be on the developing lung, the lung of the child and the lung of the adult, and a long way from being able to say "yes, it does" or "no, it does not" have any such long-term effect. I do not have time to mention in detail the very important recent

studies on the effects of low levels of ozone on lung cellularity, and on the morphology of mitochondria in alveolar cells. We are close to understanding why ozone exposure enhances the mortality from bacterial aerosols - almost certainly by interference with alveolar macrophages; such data do not reinforce an attitude of equanimity in respect of present exposure levels.

Lastly, I want to turn to a brief consideration of some of the things that I think should lie behind the decision about standards required to protect the public. This perhaps is the most contentious, and in some respects, the most difficult problem. I stressed that, from my vantage point, the decision as to the acceptable safety factor in respect of ozone, is an opinion. It is not a scientific fact. I have detected a great deal of confusion because people have tried to look at it as if it were, indeed, a scientific fact, whereas it is nothing more than an opinion. My opinion as to what safety factor is required to protect the normal population against the effect of ozone is an opinion. It may be an opinion influenced by all sorts of things. It has to be conditioned by a careful survey of all the available data. But it remains just that, and I am very insistent that scientists must not pretend that it is anything else; and the legislators must depend on an acceptance of what they think are the most reliable opinions as to what the safety factors ought to be. I will bring this to a focus by pointing out that we can grade the effects of oxidant air pollution in all sorts of ways, going from the least, to things that are still surmises. We don't know that we have increased hospitalization, for instance; we don't think we have increased lung cancer as an effect. But there are demonstrable adverse effects. Should we allow an observed decrement in

pulmonary function in school children after outdoor exercise on at least 30 days each year? And if you say that is acceptable, I immediately ask you "is it acceptable for 90 days?"

One reason that I am not at all inclined to brush off these effects on pulmonary function is that recovery from ozone is not a quick process. Figure 4 shows results of ozone exposure, together with continued measurements during the recovery period. It is quite normal for the pulmonary function test not to have returned to control values two hours after the exposure has ceased. This is telling us something about the way ozone is acting. It suggests to me that it is not a simple edema because that would, I think, go away more quickly. If all the ozone data showed recovery within 15 minutes of ceasing exposure, I would be somewhat happier. The fact that this recovery is rather slow makes me unhappy and hence cautious. It is very easy to say, and it is said to me occasionally, that it is really folly to be concerned in a general sense with a problem of this miniscule dimension. There are so many other much greater concerns that we should keep a sense of proportion and not be lured into thinking, when there is so much general misery, that we are doing anything useful. And, of course, it is true that in Bangkok and Teheran children face problems which have little to do with ozone. But, on the other hand, I have to say that a society only survives if it succeeds in dealing with the problems it has generated for itself. And in this sense we are in exactly the same situation as those who, a hundred years ago in Britain, started the first children's hospital (Charles Dickens was among those who raised the money). A special hospital for diseases of children seemed ridiculous to many people in the London of the 1830's. In other words, it is hard to

think of any advancement which has not been criticized on the general ground that somewhere else there are others whose needs are so much greater, that we should not concentrate on those which we can detect in our own society. I think that we have to take a strong position in this sense because I don't believe that human welfare would have been served if anyone had listened to similar arguments over the years. Lastly, I feel it is very necessary for me to sound a note of warning to the engineers and chemists amongst you in respect of the kind of input which I believe to be appropriate in terms of standard setting. There are some, and I have spoken with them, who are prepared to say that a transient decrement of pulmonary function, for instance, in children, or the effect on the lung elasticity of rats, etc., are so small that we can safely ignore them. This overconfidence, I find to be astonishing. It can, however, be dressed up to appear as more "scientific" or more "hardnosed" than is a more cautious approach, and hence is in some danger of being too readily accepted by those who have not critically looked at what is meant by "more scientific" in this context. The cautious approach in terms of standard setting, of treating ozone as the highly dangerous gas it is, is shared by almost everyone who is aware of its demonstrated effects and who has studied the lung over a long period of time, particularly its diseases and defenses and its aging. I would like to say that an insistence that people have to have an overt illness before we look carefully at the standards required to protect the growing lung of the child, for example, seems to me irresponsible.

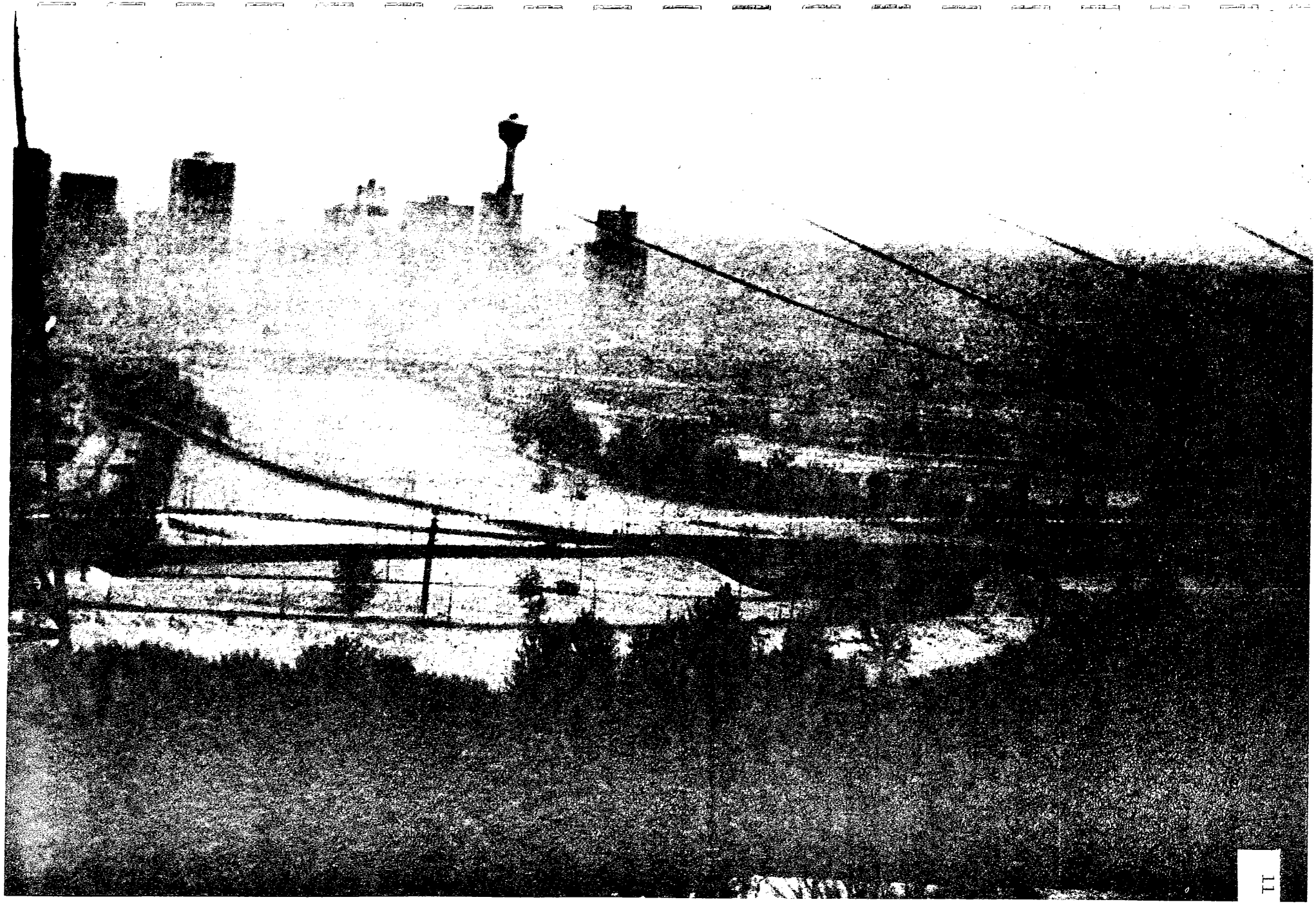


Figure 1. City of Calgary, Alberta, Canada, January, 1974 showing inversion layer.

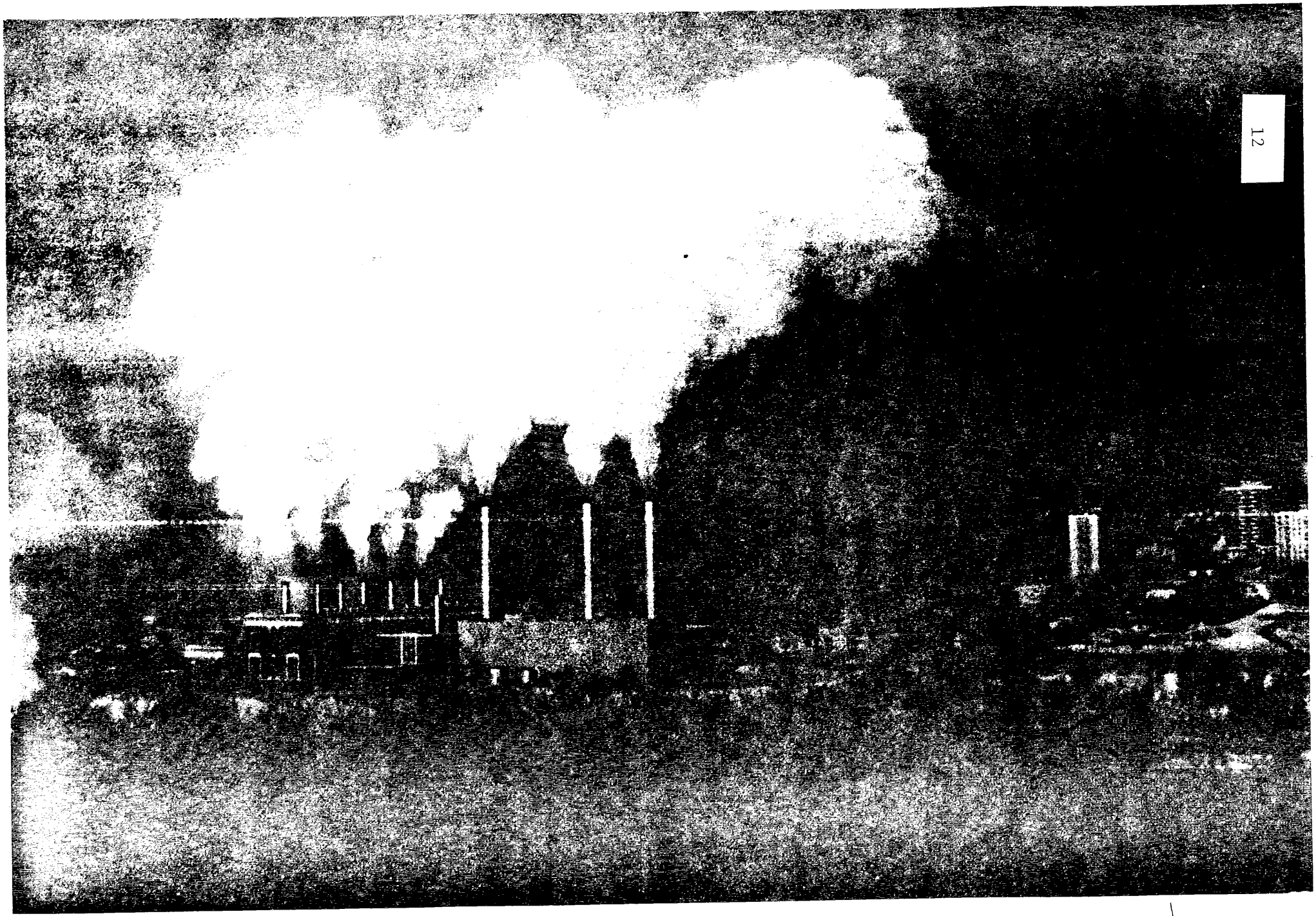


Figure 2. Electric power generation plant using natural gas near City of Edmonton, Alberta, Canada.

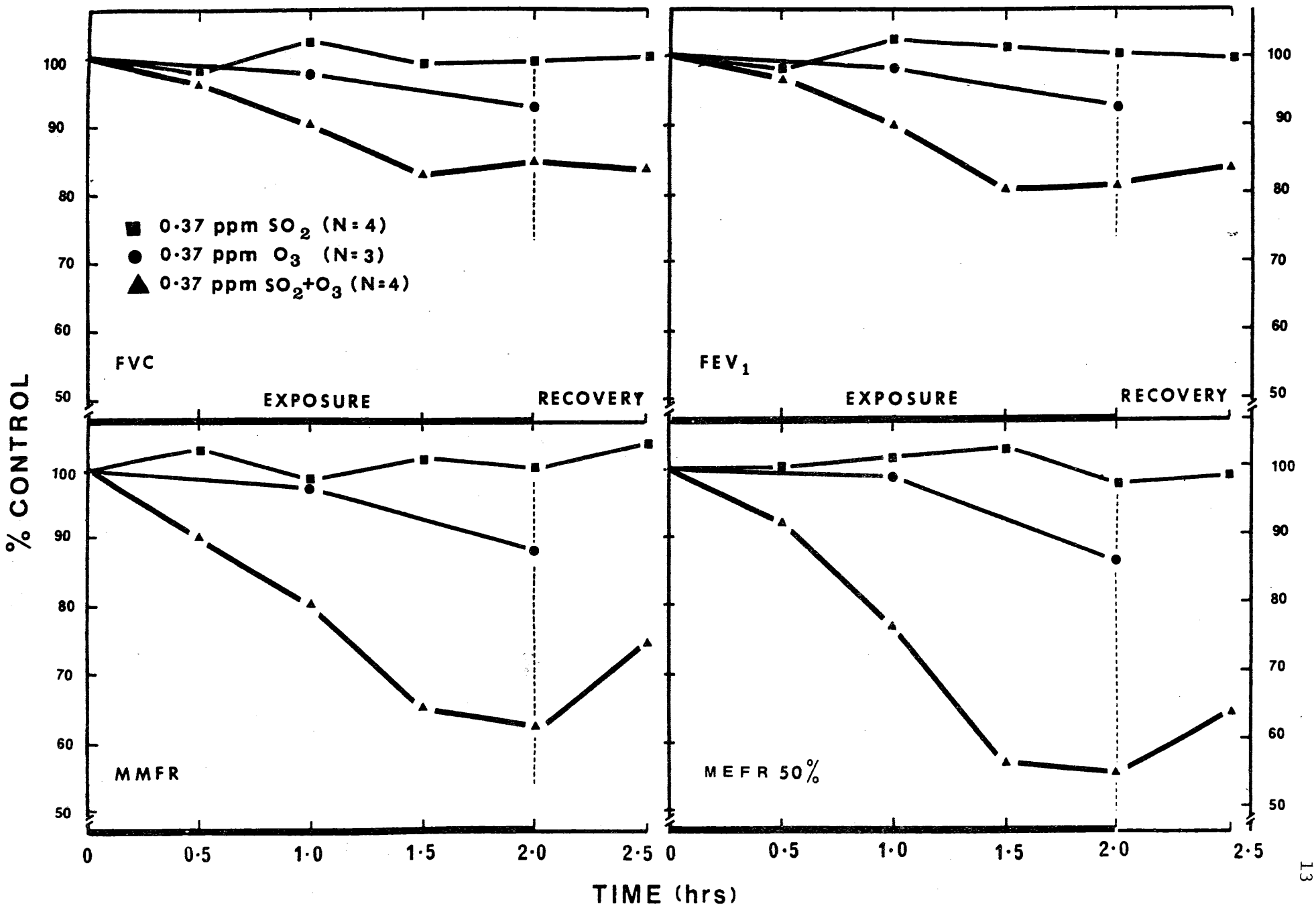


Figure 3. Effects on human lung function of 0.37 ppm ozone, 0.37 ppm SO_2, and 0.37 ppm ozone + 0.37 ppm SO_2. FVC--Forced vital capacity; FEV_1--Forced expiratory volume at 1.0 second; MMFR--Maximal mid-expiratory flow rate; MEFR 50%--Maximal expiratory flow rate at 50% vital capacity. All experiments were done with periods of light exercise alternating with rest.

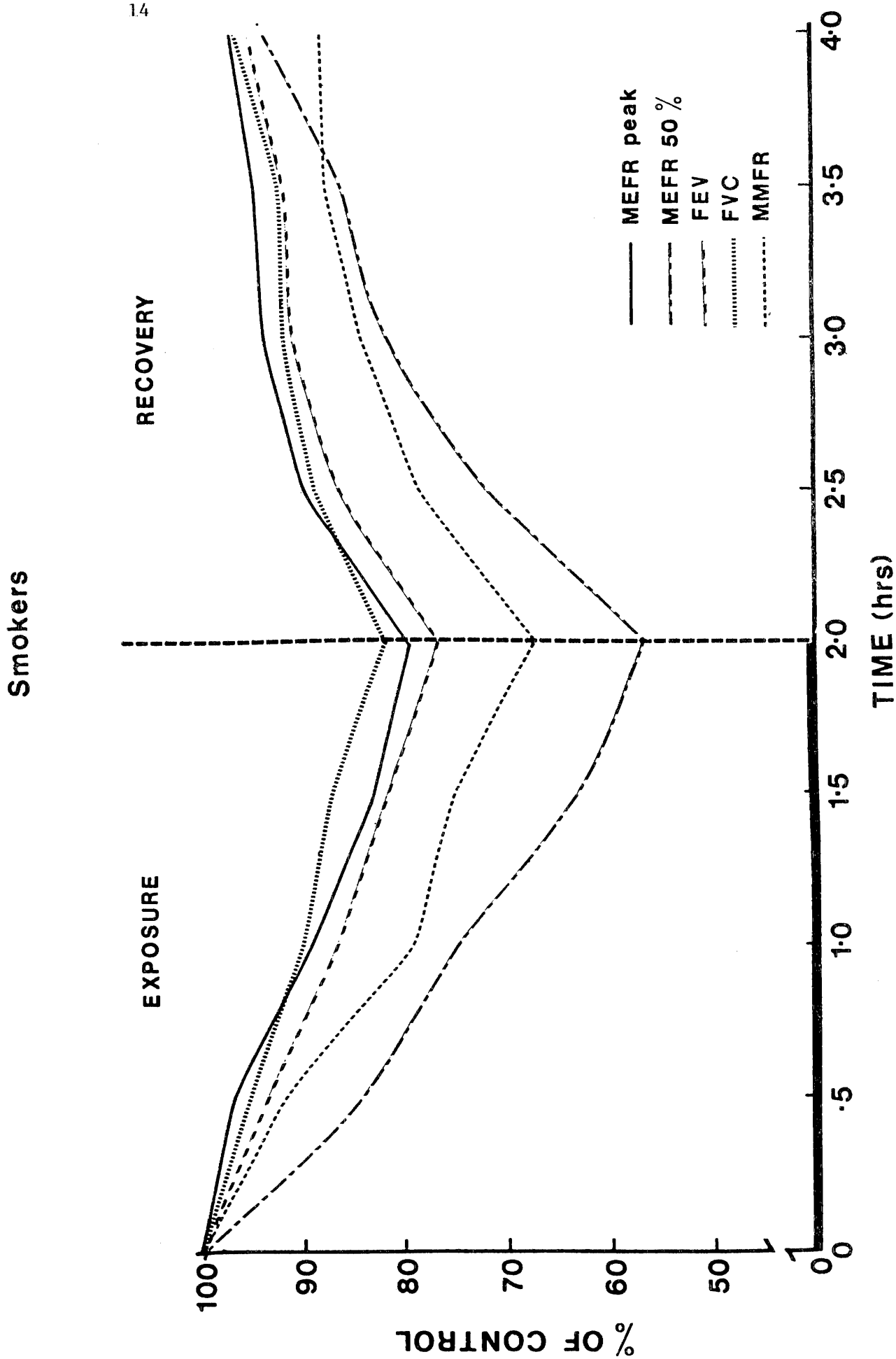


Figure 4. Time course of recovery of human lung function after exposure to 0.75 ppm ozone. Intermittent exercise performed during first two hours.

HEALTH EFFECTS OF OXIDANT EXPOSURES: A RESEARCH PROGRESS REPORT

by

John H. Knelson, Mirzda L. Peterson, George M. Goldstein,
Donald E. Gardner and Carl G. Hayes
Health Effects Research Laboratory
Environmental Protection Agency
Research Triangle Park, North Carolina

Introduction

Does the current U.S. Ambient Air Quality Standard for photochemical oxidants (160 $\mu g/m^3$ maximum one hour concentration) adequately protect, with a reasonable margin of safety, the public health? The U.S. Environmental Protection Agency has in progress a comprehensive research program designed to provide the answer to that question. Studies of humans, upon which the current standard was based, demonstrated the deleterious effects of photochemical oxidants on resistance to respiratory infections, respiratory tract and eye irritation, exacerbation of symptoms in persons with chronic lung disease, decrement in lung function, and impaired exercise performance. Studies of animals corroborated these findings and, in addition, suggested that increases in cytogenetic abnormalities and fetal wastage, as well as interference with immune mechanisms may occur with exposure to levels of ozone occurring in ambient air. Current research is continuing to explore the influence of photochemical oxidants on the respiratory disease experience and exacerbation of symptoms in healthy, as well as susceptible populations. Clinical studies are evaluating several other health parameters. Toxicological studies continue to demonstrate effects of relatively low level ozone exposure on a variety of metabolic functions.

Photochemical Oxidants: Research in Progress

- POPULATION STUDIES
  - ASTHMA ATTACK RATES
  - EXACERBATION OF SYMPTOMS
  - CHROMOSOMAL ABNORMALITIES
- CLINICAL STUDIES
  - CARDIOPULMONARY FUNCTION
  - PSYCHOPHYSIOLOGIC FUNCTION
  - CHROMOSOMAL ABNORMALITIES
  - IMMUNE STATUS
  - CARCINOMA-ASSOCIATED ANTIGENS
  - METABOLIC EFFECTS
  - EVALUATION OF COSTRESSORS
- TOXICOLOGY
  - LUNG INTEGRITY
  - IMMUNE STATUS
  - METABOLIC EFFECTS

Mechanisms of Toxicity

Ozone is known or suspected to exert its deleterious effect by a variety of mechanisms. These may be separated conveniently into two categories: those effects resulting from the action of ozone as a direct respiratory irritant, and those associated with its ability to act as a generator and propagator of free radicals or other reactive molecules such as ozonides. Some of the earliest research in ozone toxicology described alterations in the mechanical function of lung, associated with acute exposure. These were expressed as decrements in vital capacity, forced expiratory volume, flow rates, and airways resistance. Changes of this nature could be expected if ozone acted as a non-specific upper airway irritant to initiate a neurogenic response resulting in small airways constriction. Alternately, the direct action of ozone on respiratory mucosa with even a minimal resulting edema would be associated with narrowing of the small airways leading to the observed change in lung mechanical function. Although there is a paucity of data associating changes in cardiac performance with ozone exposure, one would expect a cardiac effect secondary to the change in lung function, especially in persons already suffering cardiac impairment because of pre-existing illness. Changes in small airways dynamics, as well as alterations in the mucociliary clearance mechanisms would explain at least part of the increased respiratory illness experience associated with chronic photochemical oxidant exposure in populations.

It is quite likely that ozone serves to generate and propagate free radicals and/or other reactive molecules in biologic systems. Initiation of the sequence of events resulting in formation of these reactive compounds would explain observed changes in the integrity of the organism not directly associated with the effects of ozone on the respiratory system. One would expect that free radical generation could result in changes in nucleic acid synthesis, alteration

Mechanisms of Toxicity

RESPIRATORY IRRITANT

- ALTERED LUNG MECHANICS (ACUTE)
- ASSOCIATED CARDIAC EFFECT
- ALTERED CLEARANCE

FREE RADICAL GENERATOR

- NUCLEIC ACID SYNTHESIS
  - CYTOGENETIC EFFECT
    - CARCINOGENESIS
    - MUTAGENESIS
    - TERATOGENESIS
  - IMMUNE STATUS
- PROTEIN STRUCTURE
  - ALTERED LUNG MECHANCIS (CHRONIC)
  - IMMUNE STATUS
  - METABOLIC EFFECTS
- MEMBRANE INTEGRITY
  - IMMUNE STATUS
  - METABOLIC EFFECT
  - PSYCHOPHYSIOLOGIC EFFECT
  - DIRECT CARDIAC EFFECT

in protein structure, and interference in the function of cell and organelle membranes. An interference with nucleic acid synthesis could result in cytogenetic alterations similar to those associated with carcinogenesis, mutagenesis, and teratogenesis. By the same mechanism one could anticipate an alteration in cellular immune status. Oxidation of protein cross-linkage would result in the observed chronic effects on lung structure, as well as function. Similar changes in antibody structure would account for the postulated impairment of humoral immune status. Metabolic effects such as changes in serum enyzme levels associated with a specific organ damage could also be expected as a result of alteration in the protein structure of the cellular proteins or the enzymes themselves. It is known that ozone exposure can result in peroxidation of lipoprotein cellular membranes. Such changes might be expected to cause a decrement in cellular immune status such as that associated with chemotactic or phagocytic activity. Alterations in cell membrane function resulting in leakage of intracellular contents could be expected to result in elevation of certain serum enzyme levels. Oxidation of red blood cell membranes may interfere with oxygen carrying capacity, which in turn could impair the central nervous system, as well as cardiac performance.

With these possible mechanisms of toxicity in mind, we have designed a research strategy to evaluate the biological significance for man of repeated exposures to low levels of oxidant air pollution.

Planning a Research Strategy

The design of a coherent program to evaluate the human health effect of environmental factors is a complex undertaking. Four of the most important aspects of such a program, however, are the following:

(1) Definition of the most likely clinical changes associated with known or suspected mechanisms of toxicity.

(2) Description of the concentration and distribution of the toxic environmental agent in the biosphere.

(3) Description of the nature, size, and distribution of the population at risk.

(4) Evaluation of potential co-stressors.

The usual techniques of epidemiology, clinical studies, and basic toxicology permit, in varying degrees, an approach to these four aspects of environmental health effects research. The strengths and limitations of the three disciplines are well understood and appreciated. Somewhat less thought, however, has been given to the difficulties encountered in deriving an exposure density function for populations. The basic question is "How many people in various susceptibility categories are exposed to what levels of which pollutants and for how long?" Answering this question is essential to the development of a meaningful human damage function.

The United States Environmental Protection Agency is approaching this problem by developing technology in four separate areas:

(1) Environmental monitoring

(2) Regional modelling

(3) Clinical research in controlled environmental laboratories

(4) Application of clinical research methods to population studies

Improved air monitoring has been achieved through use of more accurate and specific sensors capable of providing measurements with very short averaging times. The use of such principles as chemiluminescence has made possible the development of semi-automated monitoring stations which themselves are directed and calibrated by small on-board computers. These monitoring stations are sited in selected urban locations throughout the United States and feed their data by telephone lines into a command central computer located in our research

headquarters in North Carolina. Thus the air quality of regions in which we are conducting health studies can be monitored constantly.

Such monitoring, however sophisticated it is, does not give adequate dose data for the recipient population. Meteorologic conditions cause constantly varying pollutant isopleths around the monitoring stations. Use of dispersion models provides a method for better estimation of local pollutant concentrations. These models are validated or improved by mobile monitoring stations that can make many intermittent analyses throughout a region, as well as by monitoring grids established specifically for that purpose. Because people do not remain stationary in a given region, activity profiles are being developed to model their movements through pollutant gradients. Classic methods of epidemiology, used with these estimates of population dose, provide the data base for our Community Health and Environmental Surveillance System - a systematic assessment of associations between environmental factors and public health. In addition, targeted epidemiologic studies designed to answer specific questions are conducted.

Interpretation of data from population studies is fraught with many well-known difficulties. When possible, it is desirable to corroborate the epidemiologic findings with those of carefully controlled clinical studies. The standard techniques of the clinical scientist are used to assess transient and subtle changes in the health status of his subjects. Not standardized, however, are the techniques for manipulating and controlling the subjects' environment. Controlled Environmental Laboratories of relatively simple design have been in use for several years, but just coming into existence is a second generation of these laboratories which will use the same instruments to control the interior environment as are used in our cities to measure the ambient environment. Control mechanisms linked to these monitoring instruments provide the capability for programming diurnal pollutant cycles to simulate those

occurring in urban areas.

One of the principles followed in designing clinical environmental studies, is that the exposure schedule should not generate a stress in excess of that being placed on large numbers of people breathing ambient air. This principle emphasizes once more the importance of accurate and detailed air quality data.

Our current clinical studies of ozone can serve as a useful example of how air quality data are interpreted for experimental design. For example, in Figure 1 it is seen that during two months of the year in Glendora, California the daily maximum hourly average ozone concentrations equalled or exceeded 600 $\mu g/m^3$ about ten percent of the time. Thus, on the average, at least one hour each day for 36 days that year, people in Glendora were exposed to a minimum of 600 $\mu g/m^3$ of ozone. But what might have been peak and hourly concentrations just before and after the daily maximum hourly average? Figure 2 shows that for days when the maximum hourly average is 600 $\mu g/m^3$ or greater, peaks may be as much as 100 $\mu g/m^3$ higher than the maximum hourly average, but preceding and succeeding hourly concentration could be 100 $\mu g/m^3$ or more lower. An automated air monitoring system provides much more rigorous analysis of such exposure profiles for purposes of calculating population damage functions, as well as designing controlled environmental studies.

Clinical Studies

To date, most studies investigating effects of ozone in human volunteers in a controlled setting have focused on pulmonary effects. Bates and co-workers (1972,1973) demonstrated decrements in lung function after one hour and two hours of exposure to levels of ozone of 740 $\mu g/m^3$. Kerr et al (1975) exposed healthy volunteers to 1000 $\mu g/m^3$ ozone for six hours and demonstrated decrements in certain parameters of lung function after four hours of exposure to this contration.

FIGURE 1

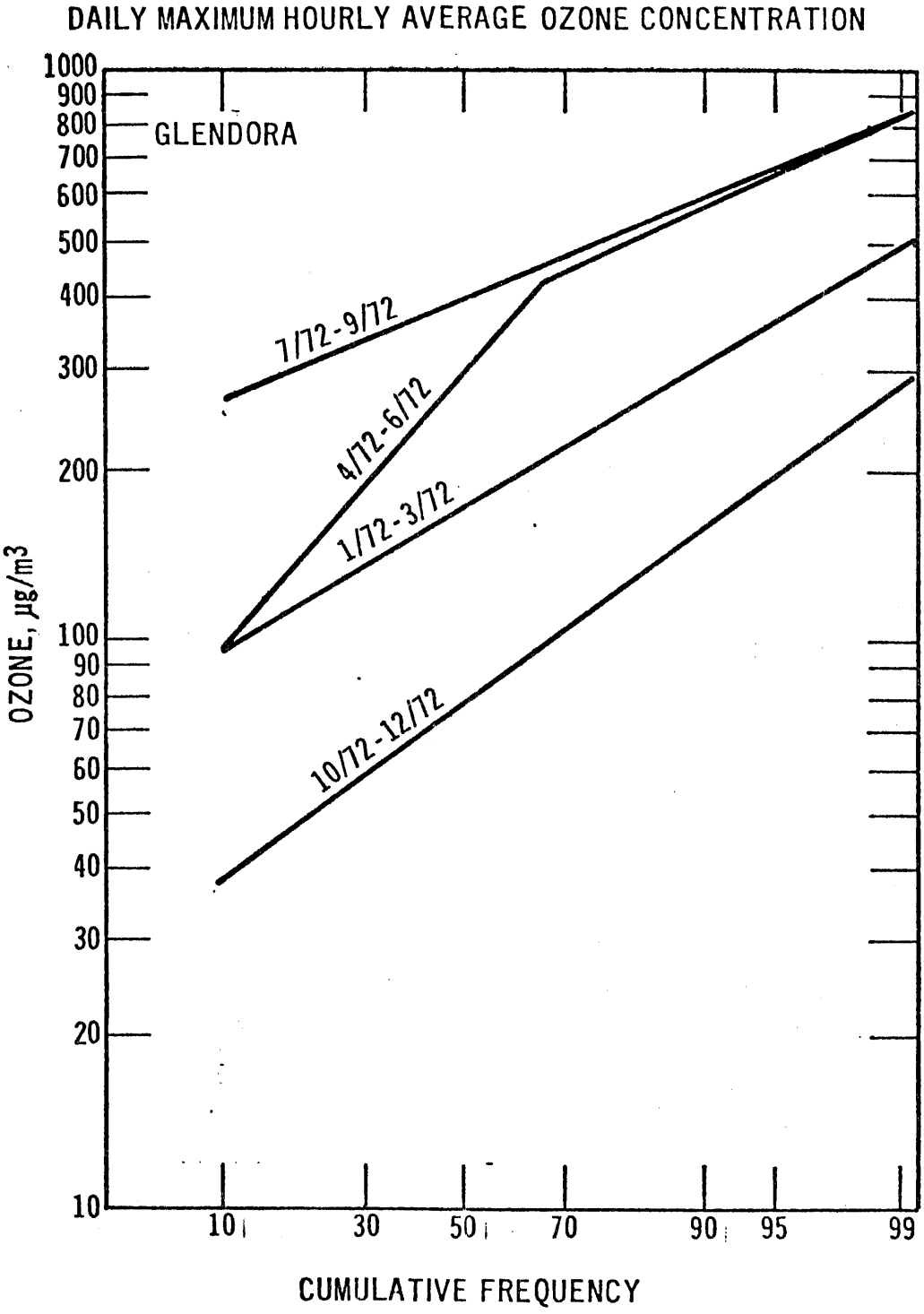
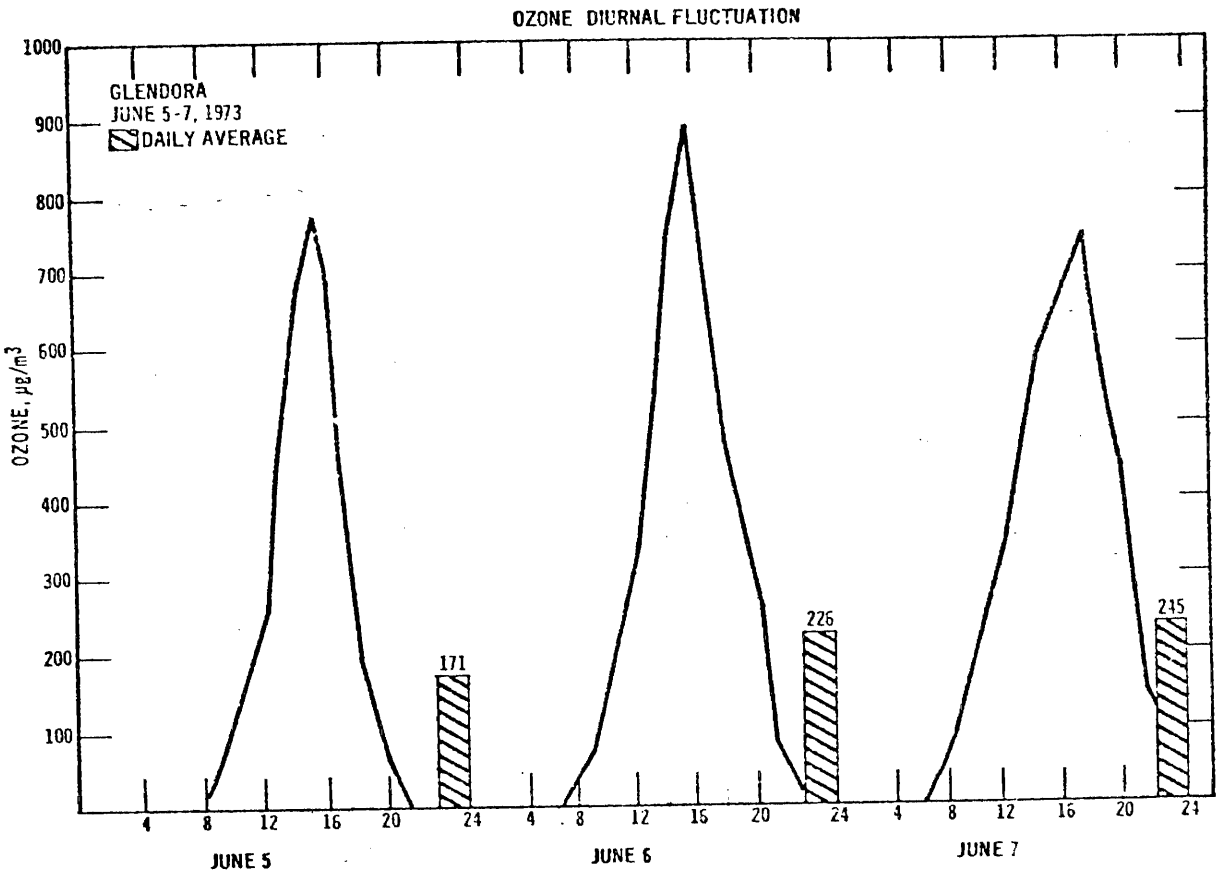

DAILY MAXIMUM HOURLY AVERAGE OZONE CONCENTRATION
GLENDORA
7/72-9/72
4/72-6/72
1/72-3/72
10/72-12/72
OZONE, µg/m3
1000
900
800
700
600
500
400
300
200
100
90
80
70
60
50
40
30
20
10
10
30
50
70
90
95
99
CUMULATIVE FREQUENCY

FIGURE 2


OZONE DIURNAL FLUCTUATION
GLENDORA
JUNE 5-7, 1973
DAILY AVERAGE
OZONE, µg/m³
1000
900
800
700
600
500
400
300
200
100
171
226
245
4
8
12
16
20
24
JUNE 5
JUNE 6
JUNE 7

There exists a body of information demonstrating ozone effects on other organ systems in animals. Chinese hamsters, following a five hour 400 $\mu g/m^3$ exposure to ozone were found to have chromosomal abnormalities in circulatory lymphocytes not found in a control group. These results confirmed previous findings of increased numbers of chromosomal abnormalities occurring in human cells exposed *in vitro* to 16 mg/m^3 (8 ppm or 16000 $\mu g/m^3$) for 5 to 10 minutes. Other animal studies have found that ozone exerts an effect on phagocytic ability of pulmonary alveolar macrophages, after three hour exposure to 1340 $\mu g/m^3$ ozone. Further studies demonstrated increased susceptibility to respiratory infections in mice and hamsters following exposures from 2.6 to 8.8 mg/m^3 ozone.

Proceeding from this data base we initiated experiments to evaluate the effects of ozone on:

(1) Lung function

(2) Chromosomal abnormalities

(3) Lymphocyte transformation

(4) Neutrophil adherence and chemotaxis

(5) Neutrophil phagocytic and killing rates

(6) Carcinoma - associated antigen titres

(7) Serum enzyme levels.

These studies are still in progress but some interesting preliminary results have been obtained.

Males between the ages of 20-27 are given a four hour exposure to 800 $\mu g/m^3$ ozone in a precisely controlled plexiglass environmental chamber measuring 8' x 8' x 8'. This level was chosen since it closely approximates oxidant levels occurring in the Los Angeles basin during pollution episodes and was one of the significant harm levels described in the Federal Register of October 23,

1971. The experimental protocol was approved by the University of North Carolina Committee for protection of human subjects operating under existing DHEW guidelines. All subjects completed comprehensive medical history questionnaires and were examined by a physician. No subject was accepted who had a history of respiratory, allergic or cardiac illness, or who was a smoker.

Ozone is generated by flowing bottled oxygen through a silent arc ozonator. Accurately calibrated chemiluminescence analyzers, currently used by EPA CHAMP stations monitor the ozone level in the chamber. A feedback system from the analyzer controls oxygen flow through the ozonator, and thereby keeps the ozone level within very narrow limits. Ozone levels are equal and constant in all parts of the chamber and during the exposures have been within the limits of 700 to 840 $\mu g/m^3$ at all times. The temperature is maintained between 70-76°F and relative humidity at 40-60%.

During the ozone exposure the subjects are seated in the chamber except for two exercise periods. At the one and three hour points each subject exercises on a bicycle ergometer for 15 minutes at 700 kg-meters, a level sufficient to increase minute ventilation fourfold. ECG is continuously recorded at rest and during exercise. At the two hour point subjects are removed from the chamber for approximately 15 minutes for pulmonary function testing.

The parameters measured include forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), maximal mid expiratory flow rate (MMEF), forced expiratory reserve volume (FERV), and forced expiratory reserve volume at one second ($FERV_1$). In addition, flow volume tracings are recorded and measurements of airway resistance and thoracic gas volume are made using a body plethysmograph.

Spirometry is performed with a 12 liter low resistance, dry-seal rolling

spirometer (CPI, model 220). Paper tracings are recorded on a X-Y plotter. Simultaneously the differentiated volume signal from the spirometer is displayed on the Y asix of a Tektronix 564 B storage oscilloscope, the volume being displayed on the X asis thus giving the flow-volume trace. Polaroid photographs are taken for analysis of the tracing. Three forced vital capacity maneuvers are made at each testing point, and the maximum vital capacity is later analyzed. The flow-volume tracing showing the highest peak-flow rate is chosen for complete analysis. Airway resistance and thoracic gas volume are measured in a constant displacement body plethysmorgraph (CPI #1100), using the method of Dubois, et. al. (1956).

Subjects participate in a control session breathing ambient Chapel Hill air in the chamber three days prior to the ozone exposure. Before the control session the subjects are trained in the performance of the pulmonary function studies. The four hour control session is similar in all respects to the subsequent ozone exposure. Control and ozone exposures take place from 9AM - 1:15PM for all subjects. Baseline pulmonary function measurements are performed before entering the chamber and after two and four hours of exposure to ambient air or ambient air plus ozone. Following air and ozone sessions, subjects fill out a questionnaire describing symptoms that might result from ozone exposure. These include cough and chest discomfort, as well as sham symptoms of abdominal and joint pain that could not reasonably be associated with ozone exposure. After two hours of ozone exposure, forced vital capacity and mid-maximal expiratory flow rate decreased significantly and airways resistance increased significantly. After four hours of ozone exposure, significant decreases occurred in nearly all measures of airflow and the increase in airways resistance became more marked. Thoracic gas volume and heart rate were unaffected by the ozone exposure.

Lymphocyte transformation was studied in blood from 20 healthy, non-smoking male volunteers before and after exposure to 0.4 ppm ozone for four hours. Lymphocyte transformation is a sensitive clinical marker used to examine the functional integrity of thymus-derived T lymphocytes which participate in cell-mediated immunity (Fitzgerald, 1972). This class of lymphocytes are cytotoxic and are an important defense against tuberculosis, fungal diseases and neoplastic cells. Impaired lymphocyte transformation is found in several disease states i.e., immmune deficiency diseases, systemic infections, and after exposure to radiation and cytotoxic or antimetabolite drugs e.g., cytoxan, nitrogen mustard (Nakamura, 1974).

The technique is performed on lymphocytes separated from peripheral blood via differential sedimentation (Main and Jones, 1968). The lymphocytes are then incubated with a stimulating agent, phytohemaglutinin, for three days and then labeled with C^{14} thymidine (Fitzgerald, 1971). The phytohemaglutinin stimulates the T-cells to become metabolically active and synthesize nucleic acids. In vivo this type of stimulation is produced by exogenous antigens i.e., bacteria, viruses. The degree of lymphocyte response is measured by the amount of labeled thymidine incorporated into DNA after three days of incubation (Valentine, 1971).

Figure 3 shows the lymphocyte stimulation for the 20 subjects pre-ozone exposure, immediately after four hours of exposure to 0.4 ppm, 72 hours after exposure and two weeks after exposure. Immediately after exposure lymphocyte stimulation is significantly suppressed ($p < .01$) as analyzed by analysis of variance (Winer, 1963). At 72 hours and two weeks after exposure the results are not statistically different from control levels. No statistically detectable differences were observed in lymphocyte responses in samples taken before and immediately after a four hour exposure to air.

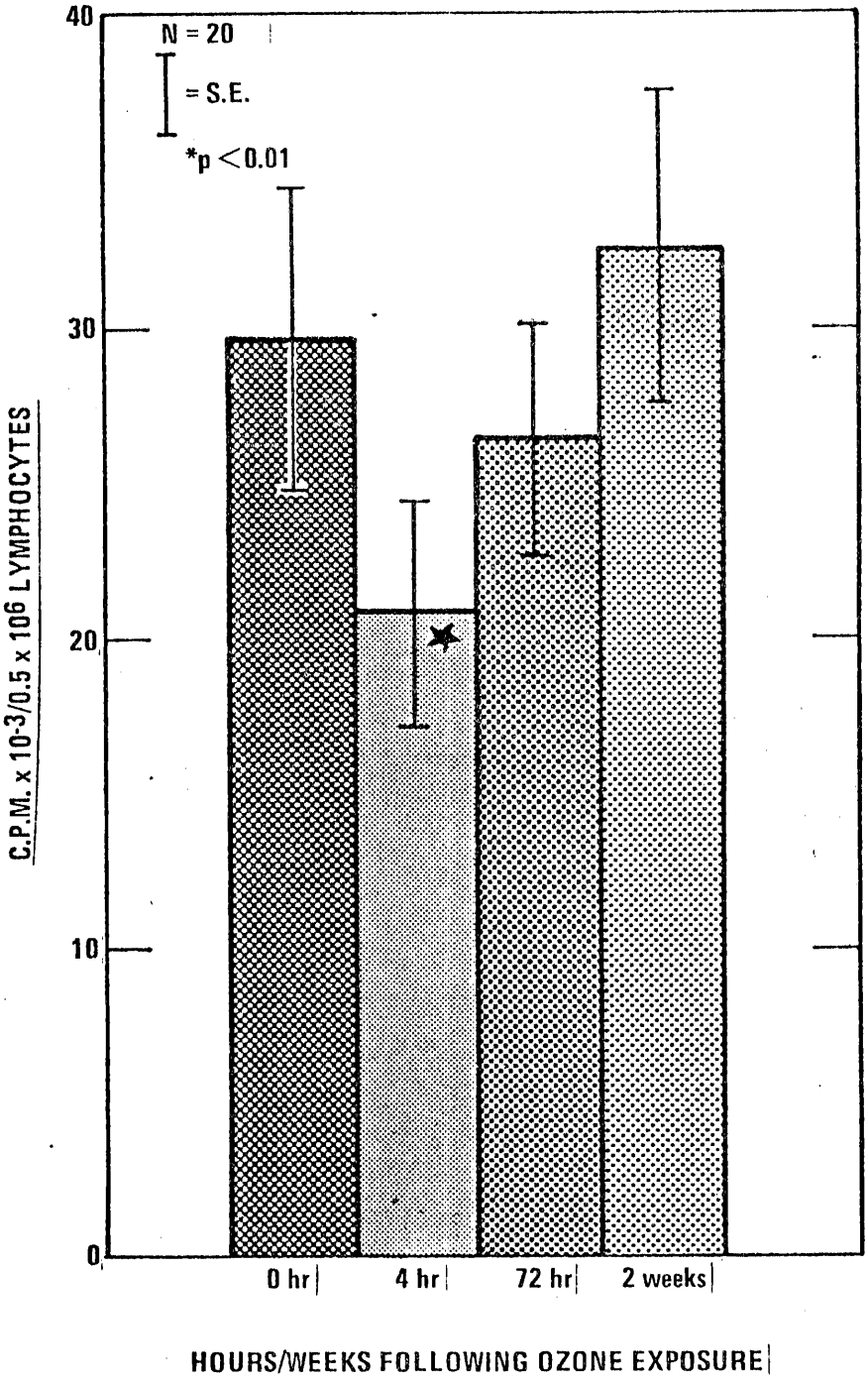


Figure 3 DNA synthesis by lymphocytes stimulated with 2 μ g/ml PHA.

The mechanism of action of ozone on lymphocytes is at present open to speculation. The existing studies suggest that ozone is radiomemetic and that the mechanism of its action may be, in part at least, through free radicals, a concept hypothesized for ionizing radiation (Brinkman, et al. 1964). It would seem then that generation and propagation of free radicals and/or other reactive molecules by ozone could result in changes in nucleic acid synthesis, alteration in protein structure, and interference in the function of cell and organelle membranes. The suppression of PHA-responsiveness of lymphocytes following exposure would then indicate that ozone had a pronounced immediate effect on the normal proliferative response of the cell, leading to blockage of DNA-synthesis, similar to the effect of UV-irradiation (Lindahl-Kiessling and Safwenberg, 1971). Since the transforming lymphocytes pass through various stages of interface activity prior to reaching DNA-synthesis, it is possible that some factor prerequisite to active cell metabolism was impaired by ozone. The obvious candidate in this context is believed to be ribonucleic acid, as the nucleic acids are considered to be the most UV-sensitive cell component. Studies in mice show that PHA stimulates primarily the more mature thymus-derived lymphocytes (Stobo and Paul, 1972). Thus, the suppressed response to PHA after the exposure may indicate that ozone impairs a more mature lymphocyte subpopulation.

Since the lymphocyte response in vitro closely approximates the normal function of cellular immunity in vivo, the continuous daily exposures to moderate concentrations of ozone may impair normal immunological functions of antibody producing B lymphocytes as a result of impairment to T cells.

The significance of the suppression of T cell response noted in this study is that: (i) if continuous exposures to ozone are shown to induce an immunosuppressed state for a significant period of time, an important

factor in carcinogenesis might be elucidated, as there is ample evidence that the immunosuppressed state is now associated with higher incidence of of malignancy (Good, 1972), (ii) immunosuppression may cause a progression of an already present tumor, (iii) immunosuppresion may enable endogenous latent infection such as tuberculosis to reactivate, and, finally (iv) immunosuppression may explain in part the relationship between chronic oxidant air pollution and influenza-like illnesses in population. Studies are in progress to assess the response of T and B lymphocytes to various mitogens and antigens and to detect membrane characteristics of lymphoid populations in humans following exposure to ozone.

Animal and in vitro studies indicating a cytogenetic effect of ozone exposure have led us to evaluate the magnitude of this effect in man. Blood taken just prior to exposure, immediately after exposure to 800 $\mu g/m^3$ ozone for four hours, at 72 hours, two weeks and one month after exposure from the same volunteers, was used to evaluate potential cytogenetic effects. Lymphocytes were cultured for 48 hours, slides made and metaphase spreads scored for chromosome aberrations. Each slide was coded so that all cytological analyses were made blindly, that is, without knowing the treatment involved. There were no statistically significant changes in chromosome morphology associated with the ozone exposure.

The phagocytic and bactericidal processes of leukocytes were studied in 20 human subjects by measuring the capability of polymononuclear neutrophiles to phagocytize and kill microorganisms of respirable size. Blood was obtained immediately prior and after a 4 hour exposure, at 72 hour and 2 weeks post exposure. We used the method of Sbarra, et al. (1965) which has been employed extensively in demonstrating the defects found in chronic granulomatous disease in children. Polymononuclear neutrophiles obtained via differential

sedimentation techniques, were allowed to interact with suspensions of Staphylococcus epidermidis, autologous serum, and Hanks' Balanced Glucose (HBG). The bacteria to phagocyte ratio of 3- 8 : 1 was chosen to produce maximal phagocytosis. After 5 minutes incubation at 37 C, the polymononuclear neutrophile suspension was added to one set of flasks, and HBG to the other. The latter served as the bacteria- serum control. The two sets were incubated in a rotating shaker (100 rpm) at 37 C. At 30 and 60 minutes intervals an aliquote was removed from each set, homogenized, diluted and plated with "pour plate" technique for the total viable bacterial count. Next, an aliquot from the experimental flask was removed, added to a portion of HBG and centrifuged. An aliquot of the supernatant was homogenized, diluted and plated for the total extracellular bacterial count. The cellular pellet was homogenized and plated for the total intracellular bacterial count. All counts were expressed as colony-forming units.

For both the phagocytic and bactericidal rates, a one-way analysis of variance with repeated measurements was used to analyze the data. For each variable, three hypotheses were used. The first hypothesis was that there was no difference in the average rate before exposure and the average rate 4 hours after exposure. For the second and third hypotheses, average rates at 72 hours and 2 weeks were used instead of the 4 hours average rate.

The 60 minutes phagocytic and bactericidal rates of leukocytes from 20 young, healthy male subjects before and following ozone exposure can be seen in Figures 4 and 5. A decrease in phagocytic ability of neutrophiles was seen immediately after ozone exposure ($p = 0.19$). The impairment was greater at 72 hours ($p < 0.05$). A significantly diminished intracellular killing was noted immediately after exposure ($p < 0.01$) and at 72 hours post exposure

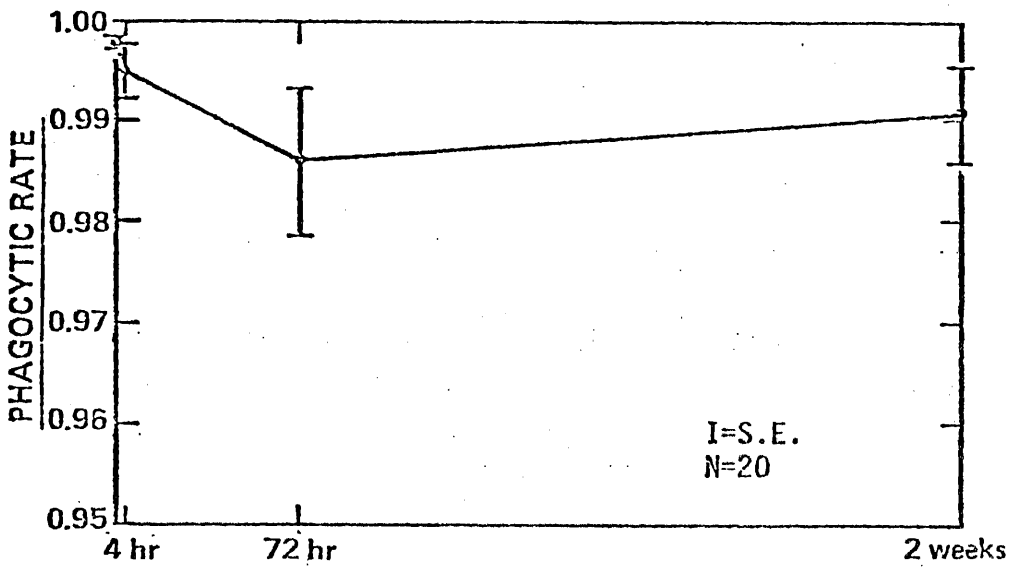


Figure 4. Phagocytic rate following ozone exposure

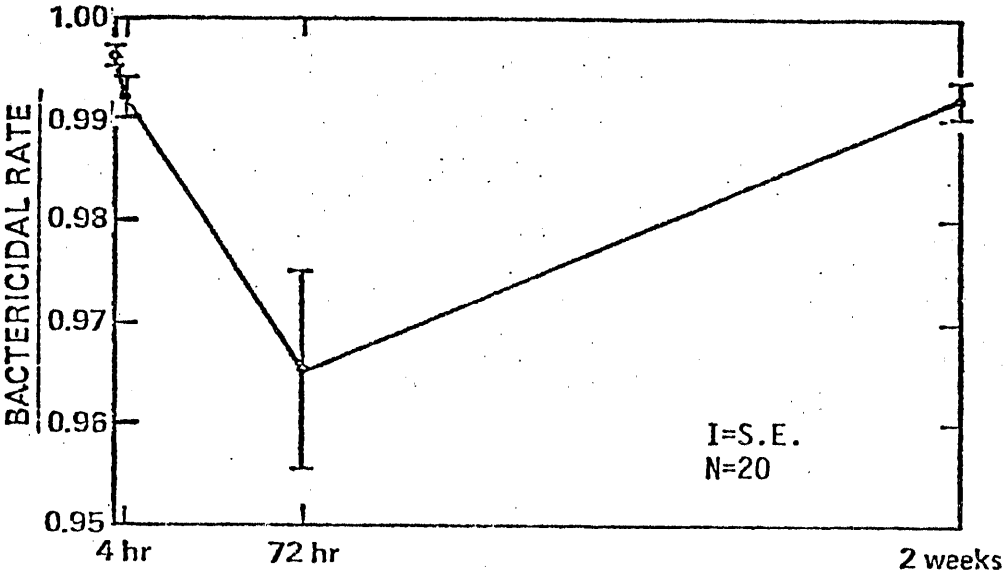


Figure 5. Bactericidal rate following ozone exposure

($p < 0.001$). Both the phagocytic and the bactericidal rates measured at 2 weeks post exposure began to return toward 0 hour values.

These findings indicate that ozone in low concentrations has an effect on cellular functions of leukocytes involved in phagocytic and bactericidal activities. Such an effect may be mediated by alterations in cell membrane, opsonization (endocytosis) functions and/or interference with intracellular enzyme synthesis. The phagocytic process may have been altered by ozone or its by-products either during the particle attachment phase between the particle and the surface of the phagocyte plasma membrane thus involving a variety of membrane receptors, or in the ingestion phase where the particle is enveloped by an invagination of the plasma membrane to form a phagocytic vacuole, or in the post phagocytic phase where the phagocytic vacuole fuses with lysosome-like granules into a phagolysosome. Normally the phagocytic process is facilitated by certain humoral factors such as opsionins among which are immunoglobulins G and M antibodies that bind to surface constituents of the microbe and heat-labile factors related to the complement and properdin systems that further augment the process. Any of these systems may have been altered. It is known that phagocytosis initiates "oxidative burst", an increased activity of the hexose monophosphate shunt pathway, and hydrogen peroxide to take part in microbicidal events. The impaired bactericidal efficiency of leukocytes would then indicate that ozone, or its by-products can interact with the intracellular processes to suppress or inhibit their microbicidal efficacy. More studies are needed towards understanding cellular systems altered by ozone. Studies are in progress to assess the effect of ozone exposure on chemotactic properties of neutrophiles.

The ability of ozone to alter organ functions remote from the site of entry was investigated by measuring organ specific enzyme indicators. It is theorized that reactive molecules, such as ozonides, and endogenous bio-active substances, originating in lung tissue, may have effects at sites remote from their origin. Murphy et al. (1964) demonstrated that inhalation of ozone caused an elevation of hepatic alkaline phosphatase.

Two liver specific enzymes, sorbital dehydrogenase and ornithine carbamyltransferase and the kidney specific enzyme, gamma-glutamyltranspeptidase were measured in subjects prior to ozone exposure, followed by measurements made at 4 hours, 3-4 days and 2-3 weeks post ozone exposure. In addition, alkaline phosphatase and glutamic oxaloacetate transaminase were measured to monitor any random changes that may occur in other tissues. Under the conditions of this exposure protocol, there was no significant difference in enzyme levels in subjects exposed to ozone when compared to their pre ozone levels (Tables 1,2,3).

Carcinoembryonic antigen (CEA) levels were shown to be elevated in the presence of carcinomas and nonmalignant diseases as well as in the plasma of smokers. Plasma CEA was measured in subjects prior to and following ozone exposure.

No significant trends were observed over the 5 week period following ozone exposure (Table 4).

The ability of ozone to alter cellular integrity, through lipid peroxidation, was measured by examining the membrane fragility of the red blood cell. Three independent methods were utilized: (1) osmotic fragility; (2) mechanical fragility and (3) hydrogen peroxide incubation. With samples from only four subjects, no discernable changes were noted (Table 1,2).

Table 1

Pre-Air Versus Post-Air Measurements[1]

Test	Means		F	Degrees of Freedom	p-value
	Pre-Air	Post-Air			
Sorbitol Dehydrogenase (12)(a)	0.94	0.71	4.3	1,11	0.06
Glutamic-Oxaloacetic Transaminase (18)(a)	15.1	14.7	0.5	1,17	0.50
Ornithine Carbonyltransferase (16)(a)	3.5	3.3	0.2	1,15	0.64
Gamma Glutamyl Transpeptidase (18)(a)	11.1	11.1	<0.0	1,17	0.98
Alkaline Phosphatase (13)(a)	89.1	86.5	0.4	1,12	0.54
RBC Osmotic Fragility (4*)(b)	12.7	9.1	41.0	1,3	<.01
RBC Mechanical Fragility (4*)(b)	7.5	10.3	20.5	1,3	0.02
RBC Peroxide Fragility (4*)(b)	0.75	0.60	0.9	1,3	0.59

*Too few measurements for a really valid test.
[1]Number in parentheses is the sample size.

a) international units
b) percent hemolysis

Table 2

Pre-Ozone Versus 4 Hours Post Versus 3-4 Days Post-Ozone Measurements [1]

Test	Means			F	Degrees of Freedom	p-value
	Pre-Ozone	4 Hours Post	3-4 Days Post			
Sorbitol Dehydrogenase (10)[a]	0.45	0.54	0.55	1.0	2,8	0.41
Glutamic-Oxaloacetic Transaminase (15)[a]	13.70	14.10	12.70	0.7	2,13	0.50
Ornithine Carbonyltransferase (10)[a]	3.91	3.53	4.11	1.2	2,8	0.36
Gamma Glutamyl Transpeptidase (15)[a]	12.47	13.80	11.73	1.7	2,13	0.22
Alkaline Phosphatase (12)[a]	82.17	77.92	84.75	2.3	2,10	0.15
RBC Osmotic Fragility (7)[b]	14.49	13.02	14.24	2.0	2,5	0.24
RBC Mechanical Fragility (7)[b]	8.95	8.33	8.08	0.3	2,5	0.74
RBC Peroxide Fragility (7)[b]	0.58	0.44	0.48	0.8	2,5	0.52

[1] Number in parentheses is the sample size.

a) international units
b) percent hemolysis

Table 3

Pre-Ozone Versus 4 Hours, 3-4 Days, and 2-3 Weeks Post-Ozone Measurements [1]

Test	Means				F	Degrees of Freedom	p-value
	Pre Ozone	4 Hours Post	3-4 Days Post	2-3 Weeks Post			
Sorbitol Dehydrogenase (8)[a]	0.53	0.65	0.65	1.14	1.3	3,5	0.36
Glutamic-Oxaloacetic Transaminase (14)[a]	13.3	14.4	12.5	13.2	1.4	3,11	0.29
Ornithine Carbonyltransferase (7)[a]	3.7	3.3	3.1	5.2	0.8	3,4	0.54
Gamma Glutamyl Transpeptidase (14)[a]	12.4	13.9	11.6	11.1	1.5	3,11	0.27
Alkaline Phosphatase (9)[a]	84.8	81.4	87.0	82.0	3.8	3,6	0.08
RBC Osmotic Fragility [b]	NO TEST						
RBC Mechanical Fragility [b]	NO TEST						
RBC Peroxide Fragility [b]	NO TEST						

Number in parentheses is the sample size.
) international units
) hemolysis

TABLE 4

Pre-Ozone Versus Post-Ozone Carcinoembryonic Antigen Measurements (ng/ml plasma)

Test	Sample Size	Pre-Ozone	Post-Ozone	F	Degrees of Freedom	p-value
Pre O_3 vs 1 day post	8	0.62	1.16	0.97	1, 7	0.64
Pre O_3 vs 3-5 days post	10	0.62	0.51	0.16	1, 9	0.70
Pre O_3 vs 2-5 weeks post	10	0.76	1.24	0.59	1, 9	0.53

It would appear from these studies that a single acute exposure to ozone at 0.4 ppm for 4 hours has minimal secondary effects on organ integrity. These experiments are preliminary in nature and biochemical investigations are continuing, so as to increase our knowledge into the biochemical effects of ozone exposure. Presently, red blood cell enzymes, plasma proteins, and erythrocyte membrane lipids are being investigated in addition to plasma enzymes. Lipid peroxidation products and endogenous bioactive substances are also being investigated.

Epidemiology

To accumulate a credible data base for approaching the question of how air quality relates to human health, the Health Effects Research Laboratory conducts a substantial epidemiologic program which addresses a variety of regulated and currently unregulated air pollutants of major concern to the Agency. Current studies which specifically address the effects of ozone or photochemical oxidants are discussed below.

Many investigations of circulating peripheral lymphocytes in populations of persons with histories of exposure to ionizing radiation have demonstated a radiation dose-cytogenetic response relationship. Other investigations have suggested that exposure to certain drugs and viral infections can cause increases in the proportion of abnormal cells in peripheral lymphocytes. Animal studies have indicated that ozone may be a particularly potent chromosomolytic agent. Because ozone is the primary constituent of photochemical oxidants, it is of interest to determine if a higher proportion of persons in populations exposed to this type of air pollution carry aberrant cells than persons not exposed. It is also of interest to determine if a continual increase in the proportion

of aberrant cells occurs and, if aberrant cells are induced, whether or not the process is reversible.

An initial effort to study these questions in the Los Angeles Basin is nearing completion. Two hundred and fifty University of Southern California freshmen, half previous Los Angeles residents and half moving into the Basin for the first time were studied during the 1974-1975 school year. Blood samples were taken as soon as possible after the students arrived for registration, after the Christmas-New Year break and at the end of the school year.

Using standard techniques, lymphocytes were cultured and 100 cells scored for a study of induced aberrations. The scoring included the assignment of each chromosome, of each cell examined, to a given group of the Denver classification, determination of the chromosome number of each cell examined, as well as noting the position on the slide of each chromosomally abnormal cell.

Analyses will include comparisons of aberration rates in the Los Angeles resident students and non Los Angeles resident students at each of the three sample times. In addition, temporal analysis of the proportion and types of abnormal chromosomes in each subject will be determined.

To develop sensible short-term air quality standards, it is necessary to have firm knowledge of human response to acute exposures to unusually high levels of ambient air pollution. Such knowledge would be adequate to answer the following questions for each major pollutant or class of pollutants: (1) Does the pollutant exert effects at concentrations down to zero, or is there a threshold which the pollution concentration must exceed before exerting effects?; (2) If there is a threshold concentration, what is its magnitude?; (3) Is there a minimum duration of exposure necessary to produce effects?; (4) If so, what is it?; and (5) Of the reported human responses to acute pollution exposure, which are based on firm and reproducible results?

A good deal of useful knowledge has emerged from epidemiologic studies of the effects of acute air pollution exposure. For instance, there is evidence of increased eye irritation and coughing at ambient oxidant concentrations above about 200 $\mu g/m^3$. Such studies usually have relied upon the judgments of subjects, whose precision in reproducing the results may be limited. Thus the knowledge gained from studies to date would be enhanced and amplified by a series of epidemiologic investigations of parameters which eliminate such judgmental decisions by the subject.

A study currently underway in the Los Angeles area was designed to minimize the possibility of subjectivity. This study examines four population groups which may represent a spectrum of vulnerability to acute oxidant exposures. At one end of the spectrum are trained runners, who may be most

resistant to adverse effects, but who may show physiologic or biochemical impairments when physically stressed. At the other end of the spectrum are documented asthmatics and chronic bronchitis patients, who may be most sensitive to changes in air pollution levels. Between these extremes are healthy outdoor workers who, because of their unusually constant outdoor exposures, may reflect acute pollution effects more readily than other healthy segments of the population.

Health data have been collected before, during, and after acute exposures to unusually high ambient air pollution levels. Specific items of health data are electrocardiograms, blood pressure, heart rate, total white cell counts, differential white cell counts (including eosinophils), blood levels of immunoglobulins, and pulmonary function tests. These tests included closing volume determinations and volume-vs.-time tracings of the entire forced vital capacity maneuver. From such tracings, the FVC, the FEV, the maximal mid-expiratory flow rate (MMEF), the maximal expiratory flow rate (MEFR), and flow-volume loops can all be extracted.

From the outdoor workers and asthmatics, measurements of pulmonary function were obtained on successive days during the season of highest pollution exposure.

Aerometric data, collected continuously throughout the period of study, are available for each study community. For all four groups under study, complete and accurate information has been collected on additional variables such as age, height, sex, race, smoking habits, socioeconomic status, and occupational exposure to respiratory irritants, which may affect the investigated parameters.

Finally, Los Angeles will be one of several major metropolitan areas in which studies of daily mortality in relation to pollutant exposures are being initiated.

Toxicology

Toxicological studies have revealed a multitude of pulmonary effects resulting from inhalation of ozone, such as pulmonary edema (Alpert, et al., 1971), proliferation of fibrous tissue (Freeman, et al., 1974), swelling and disruption of pulmonary endothelium (Bils, 1970), changes in pulmonary function (Bates, et al., 1972), impairment of pulmonary defense mechanisms (Coffin, et al., 1968 and Alpert, et al., 1971), and increased susceptibility to infection (Coffin and Gardner, 1972).

It was formerly believed that the action of ozone upon the respiratory tract was accompanied by the destruction or neutralization of the ozone and for this reason was not absorbed into the body. Nevertheless, evidence is accumulating that ozone exposure can also produce non-pulmonary effects, such as sphering of RBC (Brinkman, et al., 1964), lymphocyte chromosome aberrations (Zelac, et al., 1971), lipid peroxidation of RBC (Goldstein and Balchum, 1967), and slowed desaturation of oxyhemoglobin (Brinkman and Lamberts, 1958). However, all of these effects could have been produced by the action of ozone on these cells during passage through the pulmonary capillaries and hence may not be a result of ozone acting at some distant site. Still, others report extrapulmonary effects which cause structural changes in parathyroid gland (Atwal and Wilson, 1974), and in heart muscle as well as increased neonatal mortality (Brinkman, et al., 1964).

In our laboratory we have recently shown further evidence for the systemic action of this pollutant by measuring the disruption of the hepatic metabolism of pentobarbital following ozone inhalation. In these studies female mice (Charles River, COBS strain) were exposed to 1963 $\mu g/m^3$ for three hours daily for periods up to seven days. Control mice were exposed to clean air. Groups of ozone exposed and control mice were removed from the exposure chambers each day and injected intra-muscularly with 50 mg/kg of pentobarbital sodium. Following the injection, two parameters were measuring: (1) induction time - the time interval between injection of pentobarbital and loss of the righting reflex (when the animal remained on its back after being placed there) and (2) sleeping time - the length of time elapsed between the loss and the regaining of the righting reflex.

The induction time associated with the pentobarbital injection was not influenced by exposure to ozone. However, there was a significant increase in sleeping time associated with prior exposure to ozone. This increase was consistently manifested ($p < .05$) when pentobarbital anesthesia followed the second and third ozone exposure. No statistically significant difference in average sleeping time between control and ozone exposed mice was detected following either a single exposure or after four or more successive daily exposures.

Since after the fourth day of exposure there appeared to be no further effect on sleeping time, the question arose as to whether these animals might have developed tolerance (Coffin and Gardner, 1972). To test this hypothesis,

mice that had been exposed to 1963 $\mu g/m^3$ of ozone for 3 hours per day for 7 days were then exposed to a substantially higher concentration (9800 $\mu g/m^3$) for 3 hours and sleeping time determined. A highly statistically significant ($p < .001$) increase in sleeping time was again observed, indicating that "classical" tolerance was not a factor in these experiments.

In considering mechanisms underlying this altered response to pentobarbital, the duration of the sleeping time induced by the barbituate is primarily correlated to biotransformation of the drug in the liver (Freudenthal and Carroll, 1973). Ozone may interfere with the biosynthesis or function of the hepatic microsomal oxygenases metabolizing pentobarbital. The properties of the lung and liver oxygenases are similar (Bend, et al., 1972). Palmer, et al., 1971, found that a single ozone exposure as low as 1500 $\mu g/m^3$ for three hours reduced the activity of lung microsomal enzymes, however, no effect was observed on the liver oxygenases immediately after a single ozone exposure. Since two ozone exposures were necessary before a significant increase occurred in the sleeping time, a decline in hepatic oxygenases might not be observed until after repeated ozone exposure. Such a delay in the loss of the oxygenase could be due either to direct inactivation or to inhibition of biosynthesis.

It is interesting to speculate on the biological consequences resulting from ozone interaction with lung tissue which could produce a physiological effect at a distal target site. Although it is not likely that ozone itself could reach the microsomal enzymes of the liver

and directly alter their activity, an active ozone-induced intermediate might be produced which is then transported via the vascular system. Ozone exposure has been shown to give rise to a number of possible damaging reactions within the pulmonary tissue, viz., peroxidation of unsaturated lipids (Chow and Tappel, 1972), and oxidation loss of sulfhydryls (Stokinger, 1965; Menzel, 1970). Microsomal oxygenases are inhibited by lipid peroxidation (McCay, et al., 1971 and 1972). Menzel and his colleagues (1972) have demonstrated that certain partially oxidized species, such as fatty acid ozonides, could be responsible for the systemic effects by peroxidation of hepatic membranes. Although such reactive products have a pulmonary origin, it is plausible that they may interfere with normal function of enzymes elsewhere in the body. A systemic effect due to these reactive products could also explain the protective effects of alpha-tocopheral (vitamin E) (Menzel, et al., 1972).

Studies are now underway to demonstrate more definitively some of the mechanisms discussed. This work includes direct measurement of hepatic oxygenases and cytochrome P-450 activities after ozone exposure, and sleeping time after barbital, a barbituate not metabolized, but excreted unchanged by the kidney.

In summary, the current EPA program is providing data which confirm results of studies upon which the current standard is based and, in addition, give evidence of effects not previously described in humans. There is no new evidence to support a relaxation of the current photochemical oxidant standard. On the contrary, new research results are suggesting the current standard may not include a safety margin as adequate as previously believed.

References

ALPERT, S.M., GARDNER, D.E., HURST, D.J., LEWIS, T.R. and COFFIN, D.L. (1971). Effects of Exposure to Ozone in Defensive Mechanisms of the Lung. J. of Physiol. 31, 247-252.

ALPERT, S.M., SCHWARTZ, B.B., LEE, S.D. and LEWIS, T.R. (1971). Alveolar Protein Accumulation. Arch. Intern. Med. 128, 69-73.

ATWAL, O.S. and WILSON, T. (1974). Parathyroid Gland Changes Following Ozone Inhalation. Arch. Environ. Health. 28, 91-100.

BATES, D.V., BELL, G.M., BURNHAM, C.D., HAZUCHA, M., MANTHA, J., PENGELLY, L.D. and SILVERMAN, F. (1972). Short-term Effects of Ozone on the Lung. J. Appl. Physiol. 32, 176-181.

BATES, D.V. and M. HAZUCHA. The Short-term Effects of Air Pollutants. Assembly of Life Sciences. National Academy of Sciences - National Research Council. U. S. Government Printing Office Stock No. 5270-02105. November 1973. pp. 507-540.

BEND, J.R., HOOK, G.E.R., EASTERLING, R.E., GRAM, T.E. and FOUTS, J.R. (1972). A Comparative Study of the Hepatic and Pulmonary Mixed-Function Oxidase Systems in the Rabbit. J. Pharmocol. Exp. Ther. 183, 206-217.

BILS, R.F. (1970). Ultrastructural Alterations of Alveolar Tissue of Mice: III Ozone, Arch. Environ. Health. 20, 468-480.

BRINKMAN, R. and LAMBERTS, H.B. (1958). Ozone as a Possible Radiomimetic Gas. Nature. 181, 504-504.

BRINKMAN, R., LAMBERTS, H.B. and VENINGA, T.S. (1964). Radiomimetic Toxicity of Ozonised Air. Lancet. 1, 133-136.

CHOW, C.K. and TAPPEL, A.L. (1972). An Enzymatic Protective Mechanism Against Lipid Peroxidation Damage to Lungs of Ozone-Exposed Rats. Lipids. 7, 518-524.

COFFIN, D.L. and GARDNER, D.E. (1972). Interaction of Biological Agents and Chemical Air Pollutants. Anns. Occup. Hyg. 15, 219-235.

COFFIN, D.L. and GARDNER, D.E. (1972). The Role of Tolerance in Protection of the Lung Against Secondary Insults. Proceedings of Conference of Occupational Physicians, Ludwigshafer, Germany, April, 1972, pp. 344-364.

COFFIN, D.L., GARDNER, D.E. HOLZMAN, R.S. and WOLOCK, F.J. (1968). Influence of Ozone on Pulmonary Cells. Arch. Environ. Health. 16, 633-636.

DUBOIS, A.B., BOTELHO, S.Y. and COMROE, JR., J.H. (1956). A New Method for Measuring Airway Resistance in Man Using a Body Plethysmorgraph. J. Clinical Invest., 35, 327-335.

FITZGERALD, M.D. (1971). The establishment of a normal human population dose-response curve for lymphocytes cultured with PHA (phytohemagglutinin). Clin. Exp. Immunol. 8, 421-425.

FITZGERALD, M.G., (1972). A satisfactory quantitative test of lymphocyte response to phytohemagglutinin for the definition of normal control values and recognition of immunological defects. J. Clin. Path. 25, 163-168.

FREEMAN, G., JUHOS, L.T., FURIOSI, N.J., MUSSENDEN, R., STEPHENS, R.J. and EVANS, M.J. (1974). Pathology of Pulmonary Disease from Exposure to Interdependent Ambient Gases (Nitrogen Dioxide and Ozone). Arch. Environ. Health. 29, 203-210.

FREUDENTHAL, R.I. and CARROLL, F.I. (1973). Metabolism of Certain Commonly Used Barbiturates. Drug Metabolism Rev. 2, 265-278.

GOLDSTEIN, B.D. and BALCHUM, O.J. (1967). Effect of Ozone on Lipid Peroxidation in the Red Blood Cell. Proc. Soc. Biol. Med. 26, 356-358.

GOOD, R.A., (1972). Relations between immunity and malignancy. Proc. Nat. Acad. Sci. U.S.A., 69, 1026-1032.

KERR, H.D., KULLE, T.J., McILHANY, M.L., and AWIDERSKY, P. (1975). Effects of Ozone on Pulmonary Function in Normal Subjects, Am. Rev. of Respiratory Disease, 3, 763-773.

LINDAHL-KIESSLING, K. and SAFWENBERG, J., (1971). Inability of U.V.-irradiated lymphocyte cultures. Int. Arch. Allergy, 41, 670-678.

MAIN, R.K. and JONES, M.J., (1968). Mixed Leucocyte Interaction Involving mouse strains of strong and weak allogeneic specificities. Nature, 218, 1251-1252.

McCAY, P.B., PFEIFFER, P.M. and STIPE, W.H. (1972). Vitamin E Protection of Membrane Lipids During Electron Transport Functions. Ann. N.Y. Acad. Sci. 203, 62-73.

McCAY, P.B., POYER, J.L., PFEIFFER, P.M., MAY, H.E. and GILLIAN, J.M. (1971). A function for X-tocopherol: Stabilization of the Microsomal Membranes from Radical Attack During TPNH-Dependent Oxidation. Lipids 6, 297-306.

MENZEL, D.B., ROEHM, J.N. and LEE, S.D. (1972). Vitamin E: The Biological and Environmental Antioxidant. Agr. and Food Chem. 20, 481-486.

MURPHY, S.D., DAVIS, H.V. and ZARATZIAN, V.L. (1964). Biochemical Effects in Rats from Irritating Air Contaminants. Tox. Appl. Pharm., 6, 520-528.

NAKAMURA, R.M., et al, Immunopathology. Clinical Laboratory Concepts and Methods. Little, Brown and Company, Boston, 1974.

PALMER, M.S., SWANSON, D.H. and COFFIN, D.L. (1971). Effect of Ozone on Benzpyrene Hydroxylase Activity in the Syrian Golden Hamster. Cancer Res. 31, 730-733.

SBARRA, A.J., SHIRLEY, W., SELVARAJ R.J., McRIPLEY, R.J. and ROSENBAUM, E., (1965). The Role of the Phagocyte in Host-Parasite Interactions III. The Phagocytic Capabilities of Leukocytes from Myeloproliferative and Other Neoplastic Disorders. Cancer Res., 25, 1199-1206.

STOKINGER, H.E. (1965). Ozone Toxicology: A Review of Research and Industrial Experience. (1954-1964). Arch. Environ. Health. 10, 719-731.

STOBO, J.D. and PAUL, W.E. Functional Heterogeneity of Murine Lymphoid Cells. II. Acquisition of Mitogen Responsiveness and of Θ Antigen During the Ontogeny of Thymocytes and "T" Lymphocytes. Cell. Immunol. 4:367-380, 1972.

VALENTINE, F., Lymphocyte Transformation: The Proliferation of Human Blood Lymphocytes Stimulated by Antigen in vitro, p. 443-454. In: B.R. Bloom and P.R. Glade (ed.), In vitro methods in cell-mediated immunity. Academic Press, New York, 1971.

WINER, B.J., Statistical Principles in Experimental Design. McGraw Hill, New York, 1963.

ZELAC, R.E., CHROMROY, H.L., BOLCH, V.E., DUNAVANT, B.G. and BEVIS, H.A. (1971). Inhaled Ozone as a Mutagen: I Chromosome Aberrations Induced in Chinese Hamsters Lymphocytes. Env. Res. 4, 262-282.

EXPERIMENTAL SMOG CHAMBER STUDIES AND KINETIC COMPUTER MODELING IN ELUCIDATING CHEMICAL AND PHYSICAL TRANSFORMATIONS IN POLLUTED ATMOSPHERES

by

James N. Pitts, Jr.
Director, Statewide Air Pollution Research Center
University of California, Riverside

If we are to develop urban airshed models for control strategies, inputs from a variety of research areas are required. In general, a practical airshed model consists of an emission inventory, meteorology model, and a kinetic mechanism describing the chemistry occurring in polluted atmospheres. This presentation will focus on the work at the Statewide Air Pollution Research Center (SAPRC) and the Department of Chemistry at the University of California, Riverside, which has been centered on developing an experimentally validated model of photochemical air pollution. Our input involves results from basic kinetic and mechanistic studies as well as experimental data obtained from the SAPRC evacuable environmental chamber, all of which are being applied in our computer modeling work.

Recommendations for improving the usefulness of data obtained from smog chamber studies have been made by several modeling teams, and some of the most important are: (1) Using very pure matrix air; (2) precise control and measurement of temperature and relative humidity; (3) practical methods for cleaning the chamber; (4) accurate characterization of light intensity and spectral distribution; and (5) unambiguous and accurate analytical methods including in situ real time analysis for highly reactive species.

A new evacuable environmental chamber--solar simulator and air purification facility (Figure 1) which addresses these experimental criteria has been established at the SAPRC. The evacuable chamber is a 205 cu. ft. thermostated, cylindrical vessel constructed of FEP Teflon-lined aluminum with

quartz end windows. A 20-KW solar simulator was designed specifically for use with this chamber and provides a highly collimated beam with spectral distribution that closely matches that of sunlight.

This facility is being employed to generate a data base in which a kinetic mechanism for photochemical air pollution can be validated. Results of a typical smog chamber experiment in the SAPRC evacuable chamber is shown in Figure 2 for the irradiation of a propylene-NO-NO_2 mixture in air. The initial experimental conditions were 0.5 ppm propylene, 0.45 ppm NO and 0.05 ppm NO_2 in 760 torr of highly purified air. Figure 2 illustrates the type of chamber data obtainable for model refinement and validation in a study of HC/NO_x systems in which a representative olefin, paraffin or aromatic compound is used to study first single HC/NO_x mixtures, and then binary and tertiary hydrocarbon mixtures.

An additional level of complexity may be introduced by the addition of an aldehyde to the reaction mixture permitting investigation of the role of aldehydes as photoinitiators in the mechanism. The effect of adding 0.13 ppm formaldehyde in a butane/NO_x (2.0 and 0.5 ppm respectively) reaction is shown in Figure 3. Thus, the addition of formaldehyde to the butane/NO_x photolysis accelerated the rate of conversion of NO to NO_2 and the buildup of ozone.

Other experimental data shows the interrelationship of the basic kinetic rate constant to predicted concentrations of two products of the photolysis of the propylene/NO_x system. The computer kinetic model of Niki, Daby and Weinstock is sensitive to the value of the rate constant for the reaction OH + NO + M → HONO + M. Recent studies at the SAPRC to determine the rate constant for this reaction gave 3.2×10^9 ℓ mol^{-1} sec^{-1} for the

pseudo-second order reaction and provides a better fit of the predicted to the observed experimental data for NO_2 production than does the use of the Stuhl and Niki rate constant of 1.3×10^9 ℓ mol^{-1} sec^{-1}. An improved fit for the first two hours of the NO_2 and O_3 concentration profile has been obtained.

Examination of any of the current kinetic models indicates that modelers require more than just accurate data for O_3, NO, NO_2, and hydrocarbons. For example, the Hecht, Seinfeld and Dodge model predicts, in addition to products traditionally monitored in smog chambers, products such as nitric and nitrous acid, hydrogen peroxide, alkyl nitrite, and nitrates and alkyl peroxides and hydroperoxides. The importance of measuring these species becomes apparent from the fact that for a given set of rate constants three published mechanisms provide substantially different predictions for at least two of these species, hydrogen peroxide and nitric acid. The comparison of hydrogen peroxide predicted by the three different mechanisms is shown in Figure 4.

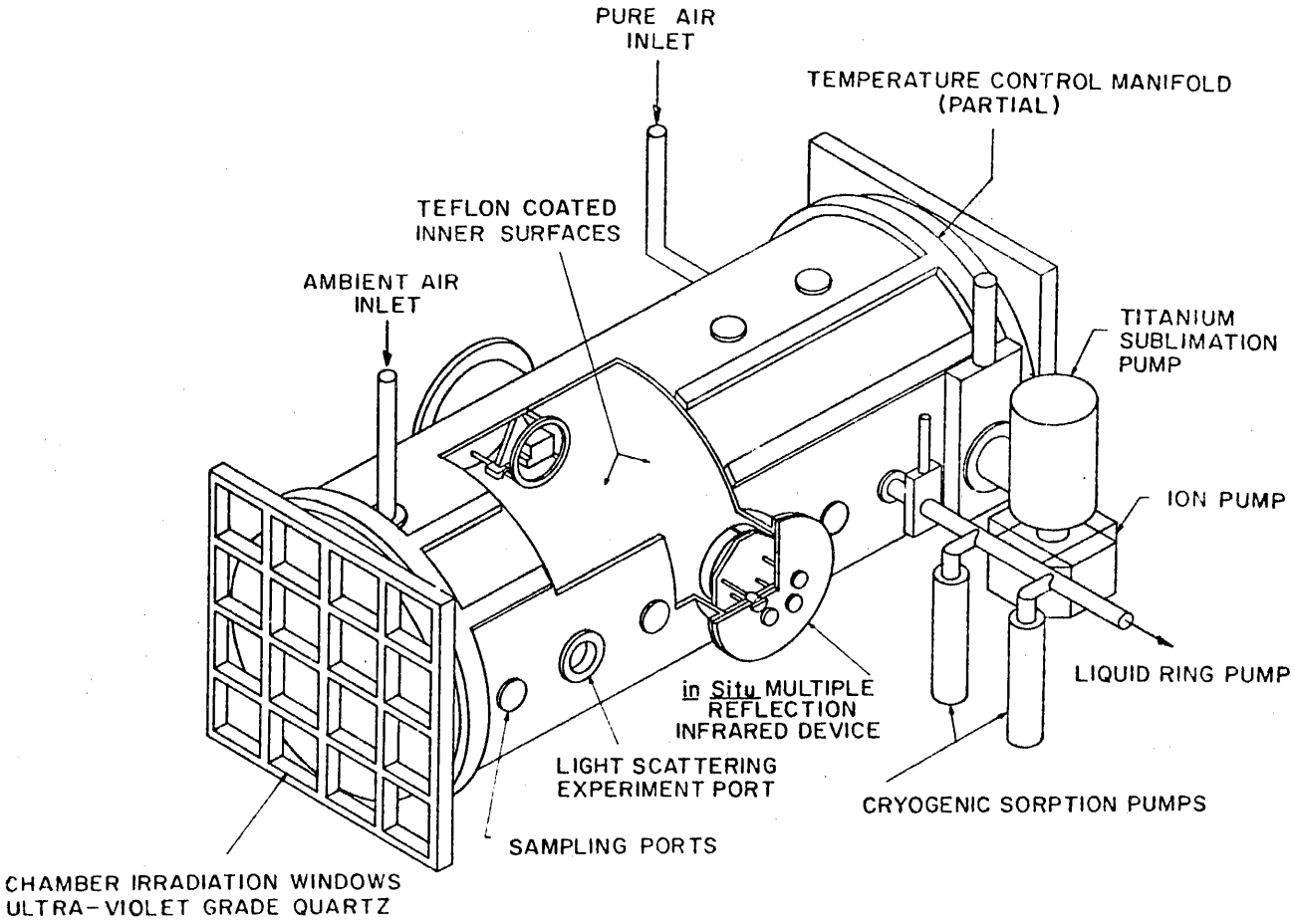
Through the use at SAPRC of a Fourier interferometer long path infrared system coupled with the evacuable chamber (Figure 5) data have been obtained for several of the less easily measured products.

Spectra obtained by this combination are shown in Figures 6 and 7. The photolysis of the mixture of NO, NO_2 and propylene in air show infrared bands assigned to PAN, ethyl nitrate, formic acid, nitrous acid, and nitric acid. Figure 8 additionally shows the results of measurements of several ozone-olefin (ethylene, propylene and cis-2-butene) reactions in the dark produced spectra of a variety of species, including ketene, and possibly peroxyformic acid and α-carbonyl hydroperoxides.

Finally, the recent oxidant measurement calibration controversy is a matter for discussion. In June, 1974, the ARB and the Los Angeles County APCD announced jointly that different methods of calibration of oxidant monitoring instruments cause oxidant measurements made by the LAAPCD to be one quarter to one-third lower than ARB measurements made at the same time and place. An outline of the history of the problem appears in CALIFORNIA AIR ENVIRONMENT, Vol. 6, No. 1, Winter 1975/76. Reported at this conference was work done in the summer and fall of 1974 at SAPRC in which the ARB potassium iodide calibration method was investigated using infrared absorption by ozone as a standard.

In order to apply the only absolute infrared absorptivity data for ozone available in the literature, a study of the dependence of the absorptivity on spectral resolution was carried out. Using the absorptivity determined for the comparatively high resolution afforded by modern infrared instruments (Figure 9) the SAPRC results for the ARB method fell in between those of the EPA and the ARB ad-hoc committee, namely that for ozone in dry air the ARB method was approximately 10% high while for wet air (50% RH) it was approximately 20% high. It should be emphasized that there is need for further studies to determine to everyone's satisfaction which method (ARB, LAAPCD, or EPA) actually gives the most accurate and reproducible values in the ambient concentration range. However, regardless of which is the best measurement technique, previous conclusions that ambient oxidant levels are highest in the eastern end of the South Coast Air Basin, are shown to be incorrect when all of the oxidant air monitoring data from the Basin through the year 1973 are placed upon a common calibration basis (see Figure 10).

Following this presentation, some results of current work from the SAPRC all-glass smog chamber are presented by Dr. George Doyle of SAPRC.



S.A.P.R.C.

EVACUABLE SMOG CHAMBER

Figure 1

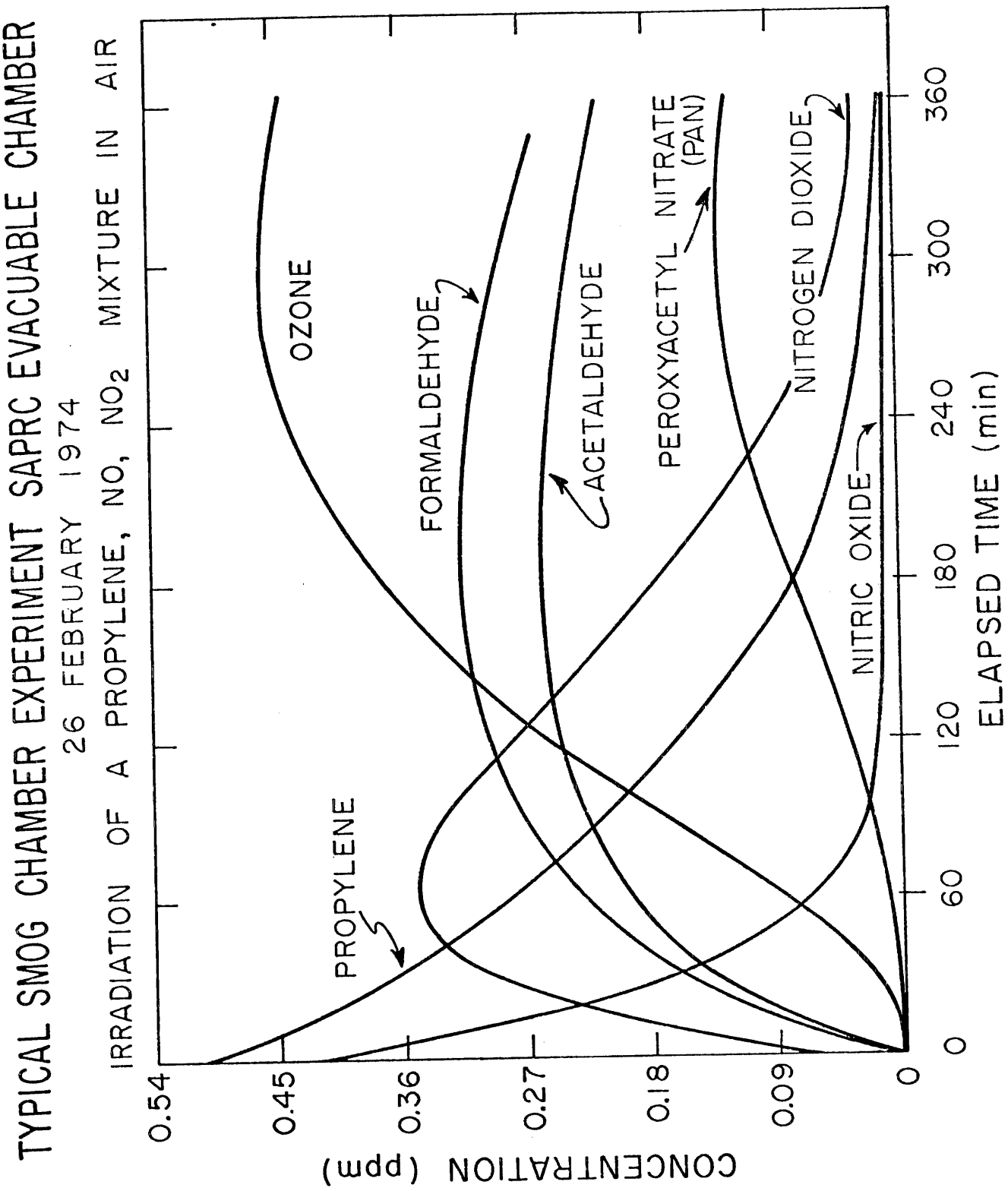

TYPICAL SMOG CHAMBER EXPERIMENT SAPRC EVACUABLE CHAMBER
26 FEBRUARY 1974
IRRADIATION OF A PROPYLENE, NO, NO_2 MIXTURE IN AIR
OZONE
FORMALDEHYDE
ACETALDEHYDE
PEROXYACETYL NITRATE (PAN)
NITROGEN DIOXIDE
NITRIC OXIDE
PROPYLENE
CONCENTRATION (ppm)
0.54
0.45
0.36
0.27
0.18
0.09
0
0
60
120
180
240
300
360
ELAPSED TIME (min)

Figure 2

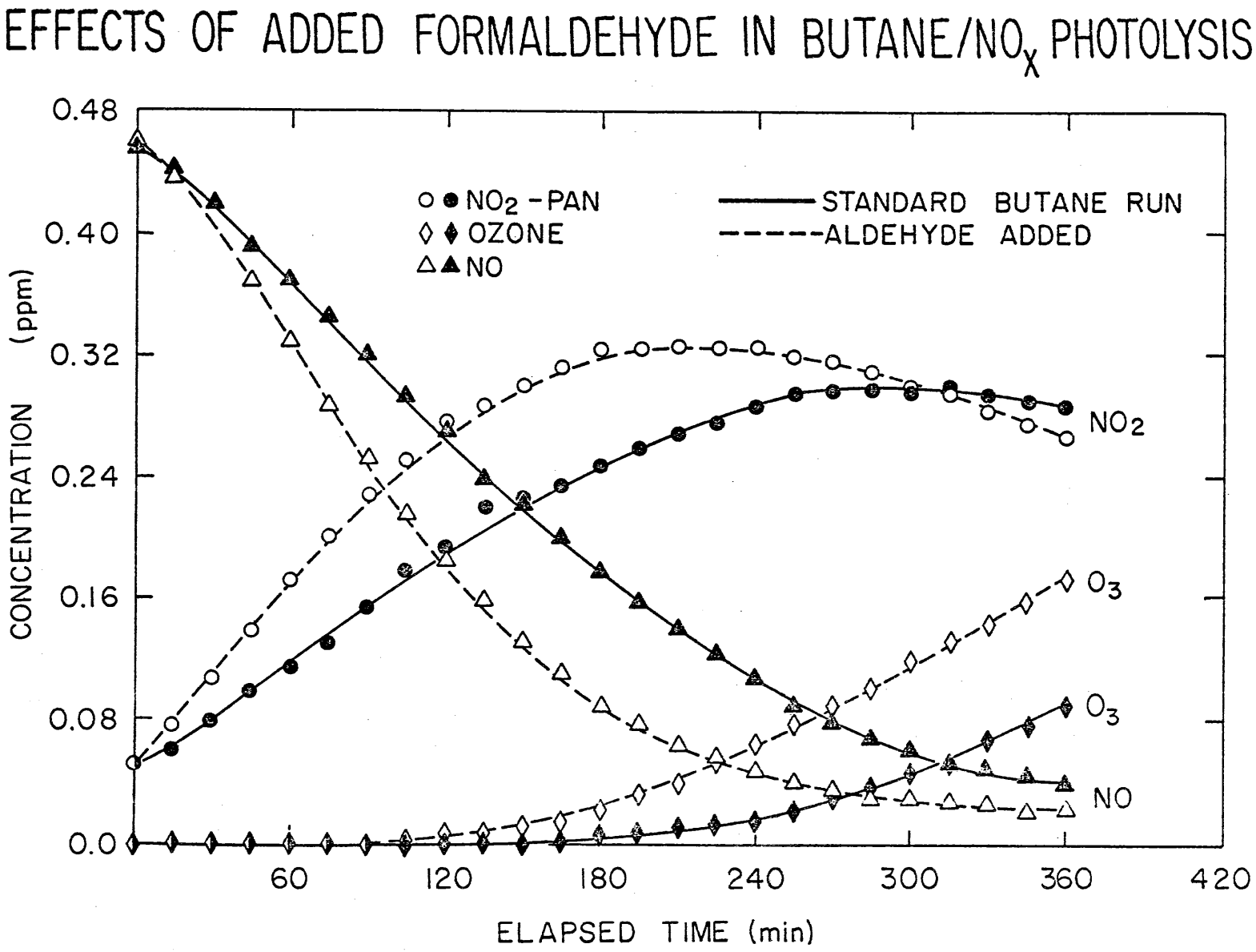

EFFECTS OF ADDED FORMALDEHYDE IN BUTANE/NOx PHOTOLYSIS
0.48
0.40
0.32
0.24
0.16
0.08
0.0
CONCENTRATION (ppm)
NO2 - PAN
OZONE
NO
STANDARD BUTANE RUN
ALDEHYDE ADDED
NO2
O3
O3
NO
60
120
180
240
300
360
420
ELAPSED TIME (min)

Figure 3

PROPYLENE/NO_x/AIR

EFFECT OF THE VALUE OF THE RATE CONSTANT OF THE REACTION

$OH + NO + (M) \rightarrow HONO + (M)$

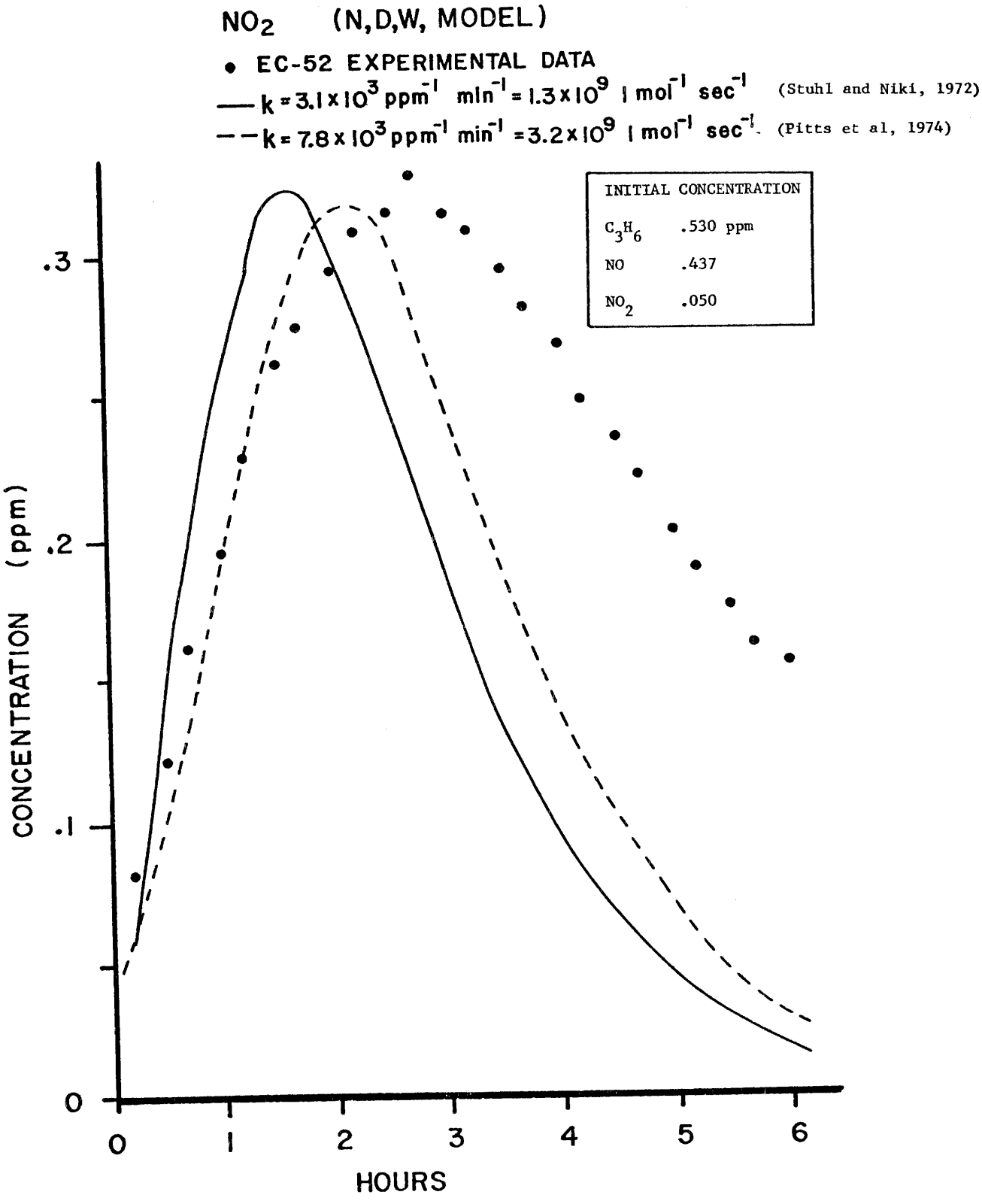


Figure 4

PROPYLENE/NO_x/AIR

EFFECT OF THE VALUE OF THE RATE CONSTANT OF THE REACTION

$OH + NO + (M) \rightarrow HONO + (M)$

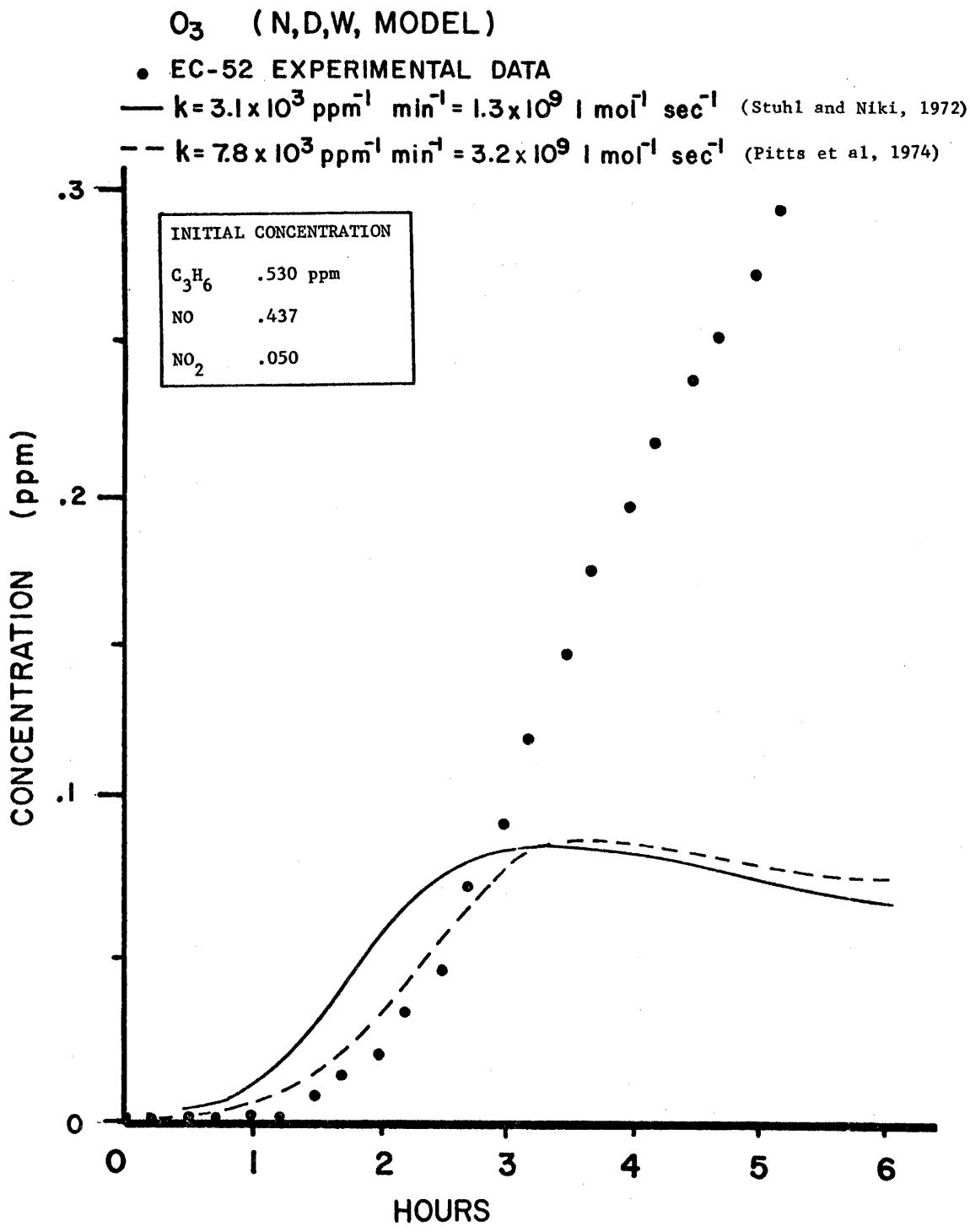


Figure 5

COMPARISON OF H_2O_2 PREDICTED BY THREE MECHANISMS

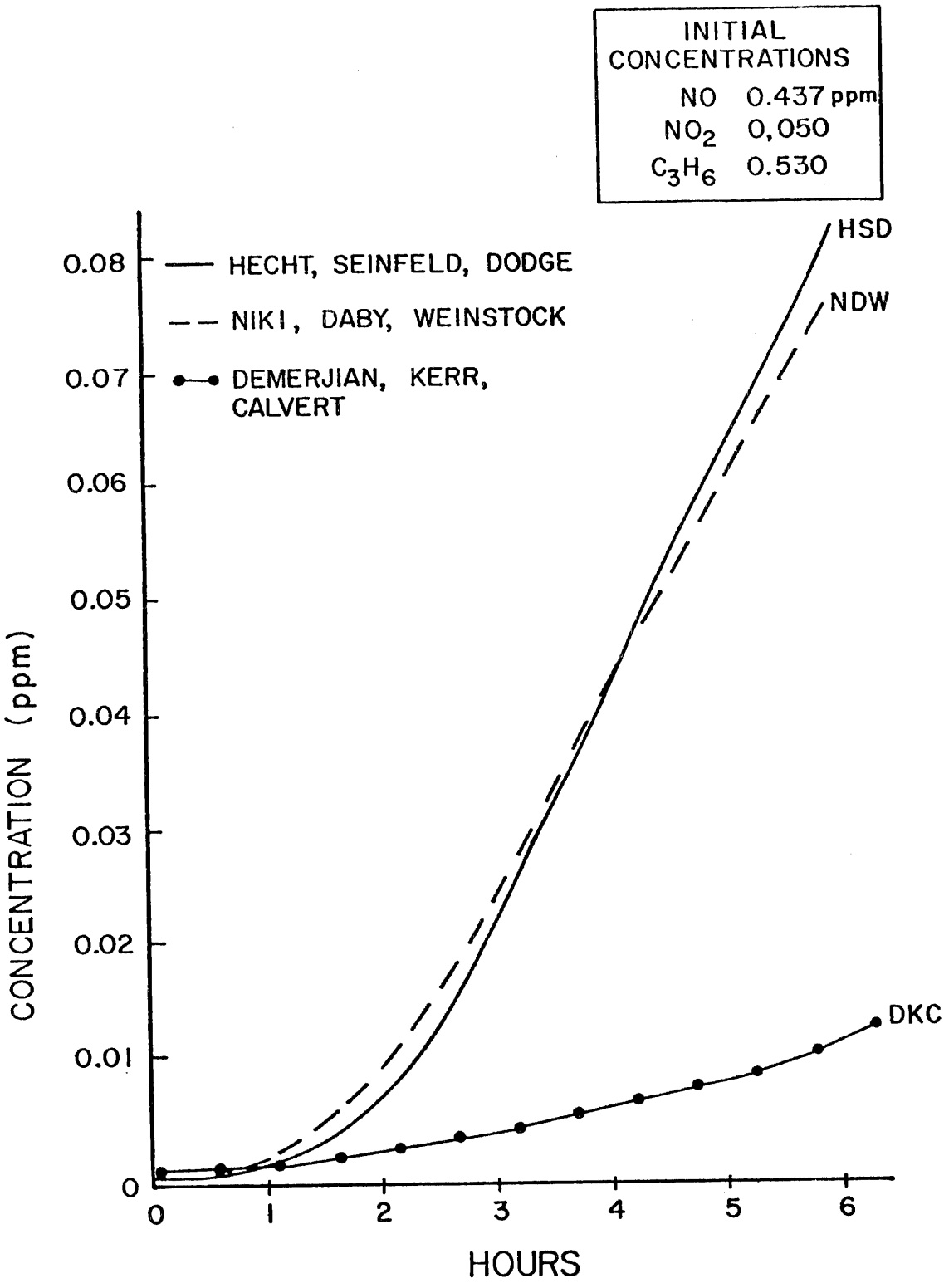


Figure 6

ENVIRONMENTAL CHAMBER AND FOURIER INFRARED INTERFEROMETER WITH IN-SITU LONG-PATH OPTICAL SYSTEM

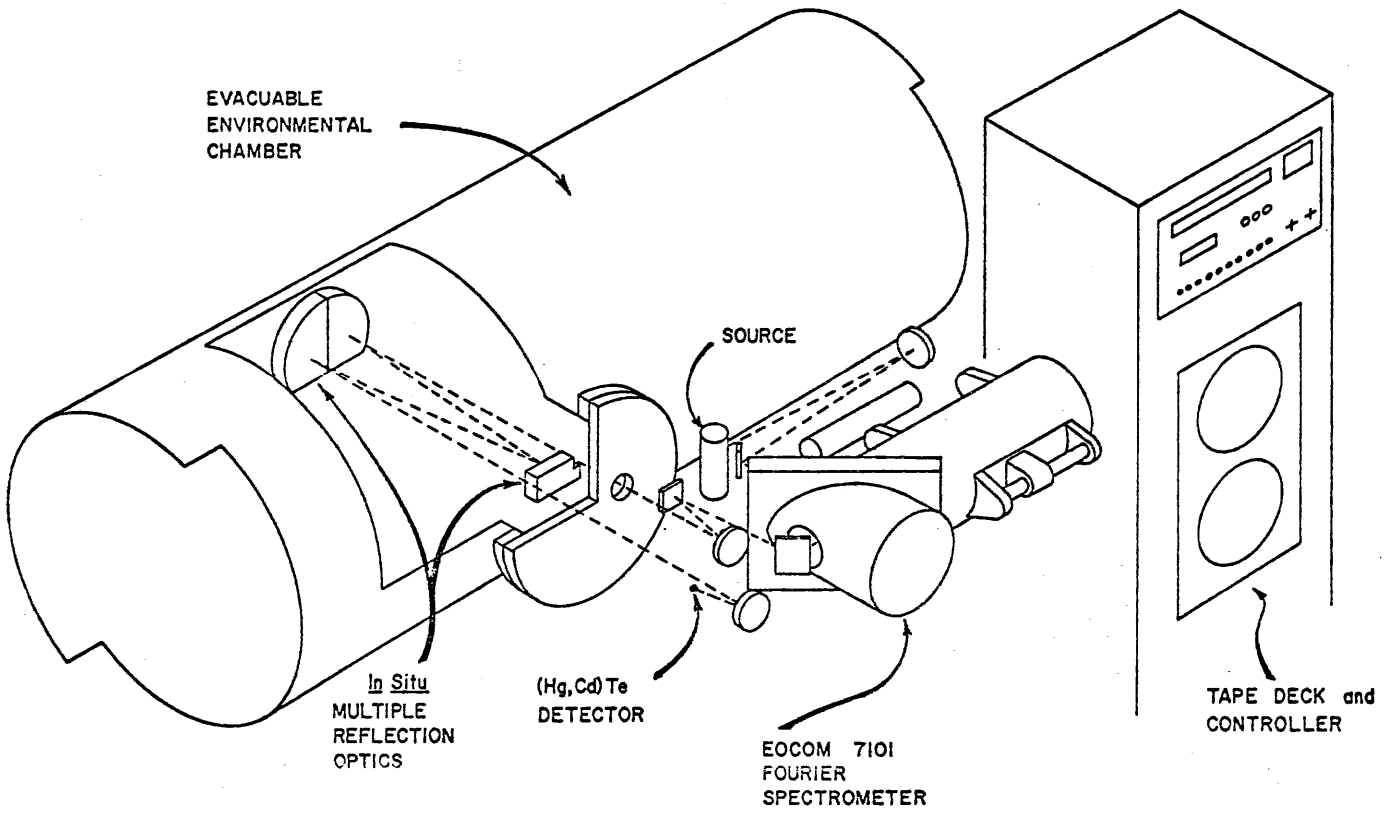
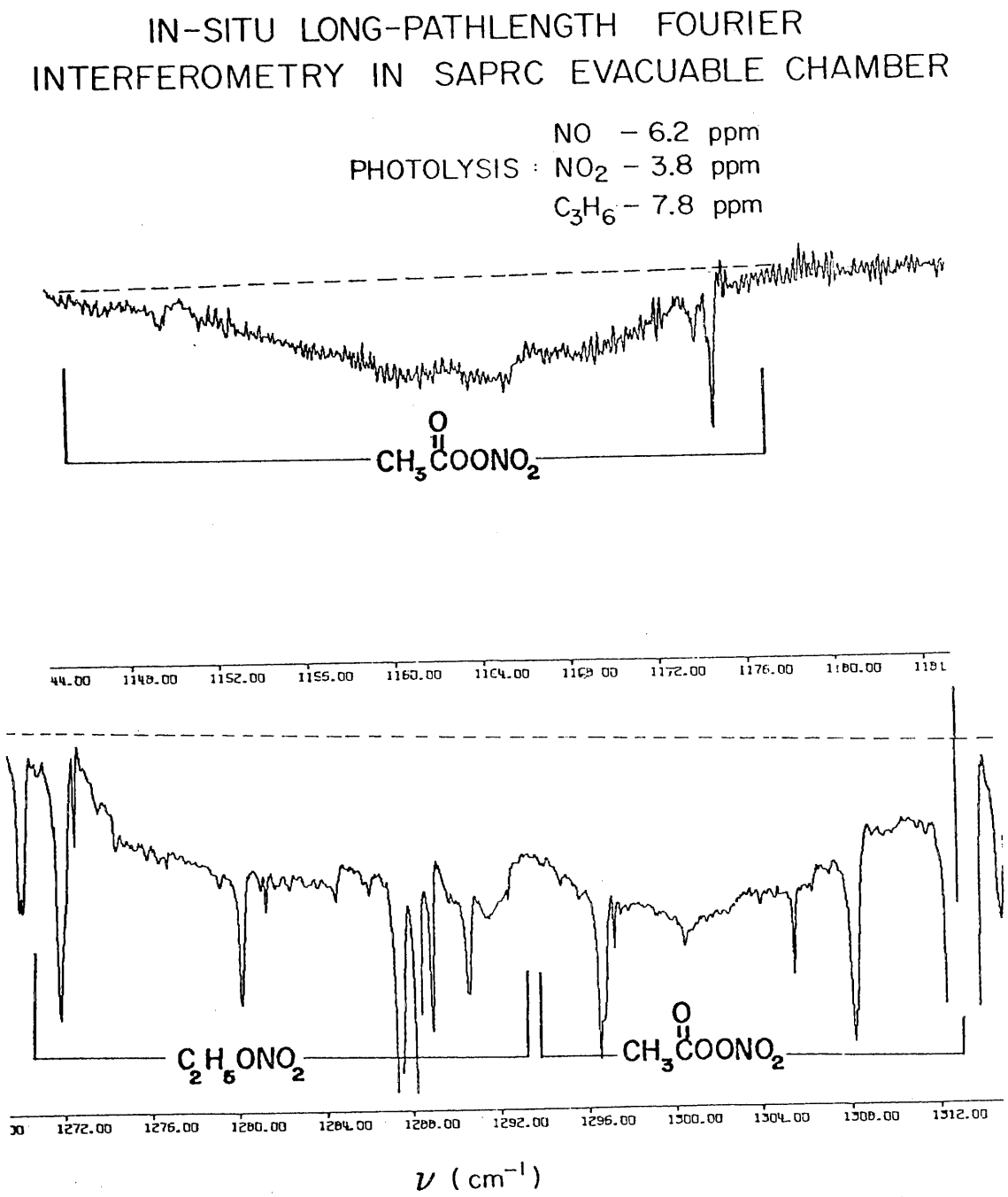
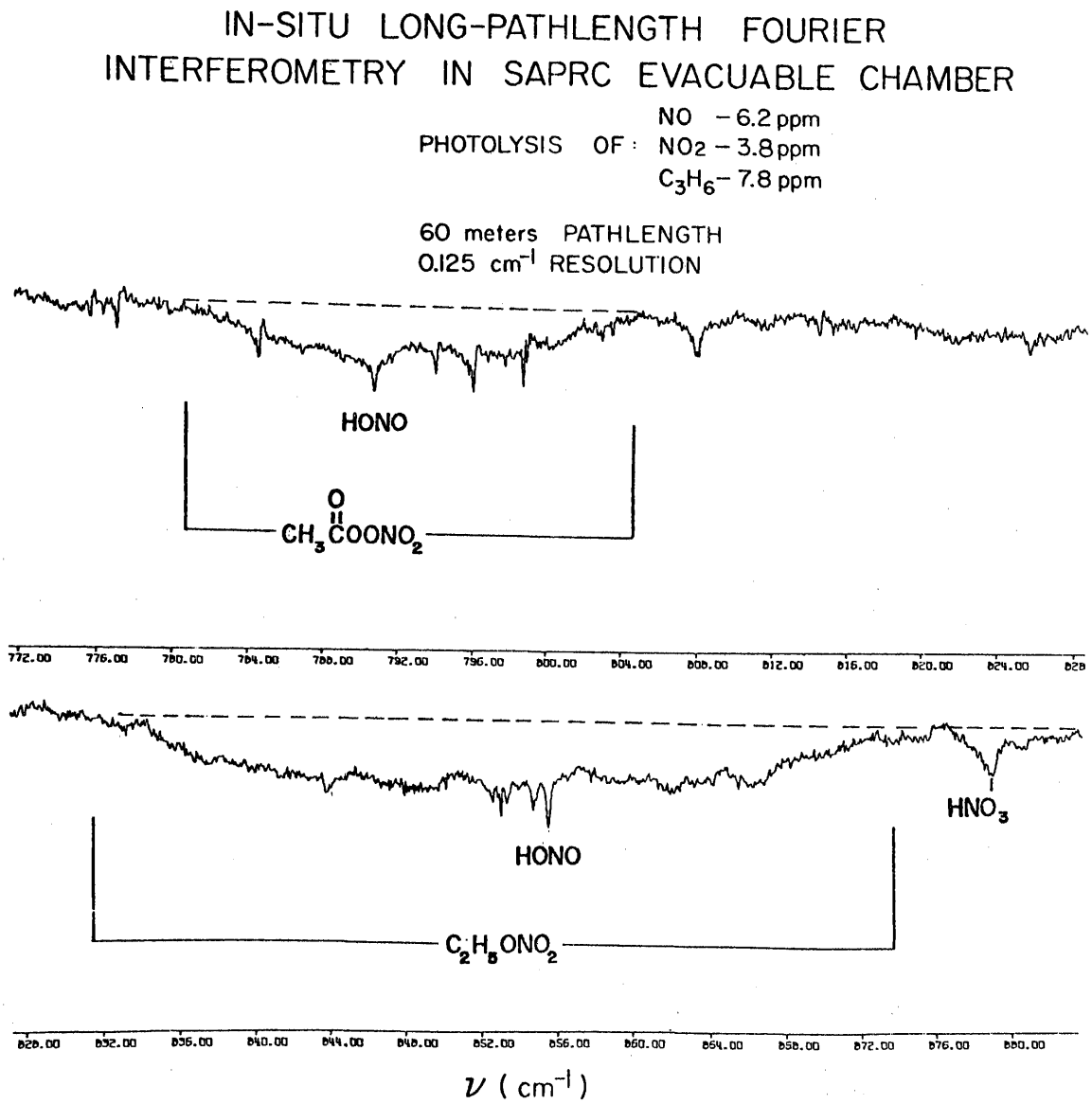


Figure 7



J.M. McAfee, A.M. Winer and J.N. Pitts, Jr. (1974)

Figure 8



J.M. McAfee, A.M. Winer and J.N. Pitts, Jr. (1974)

Figure 9

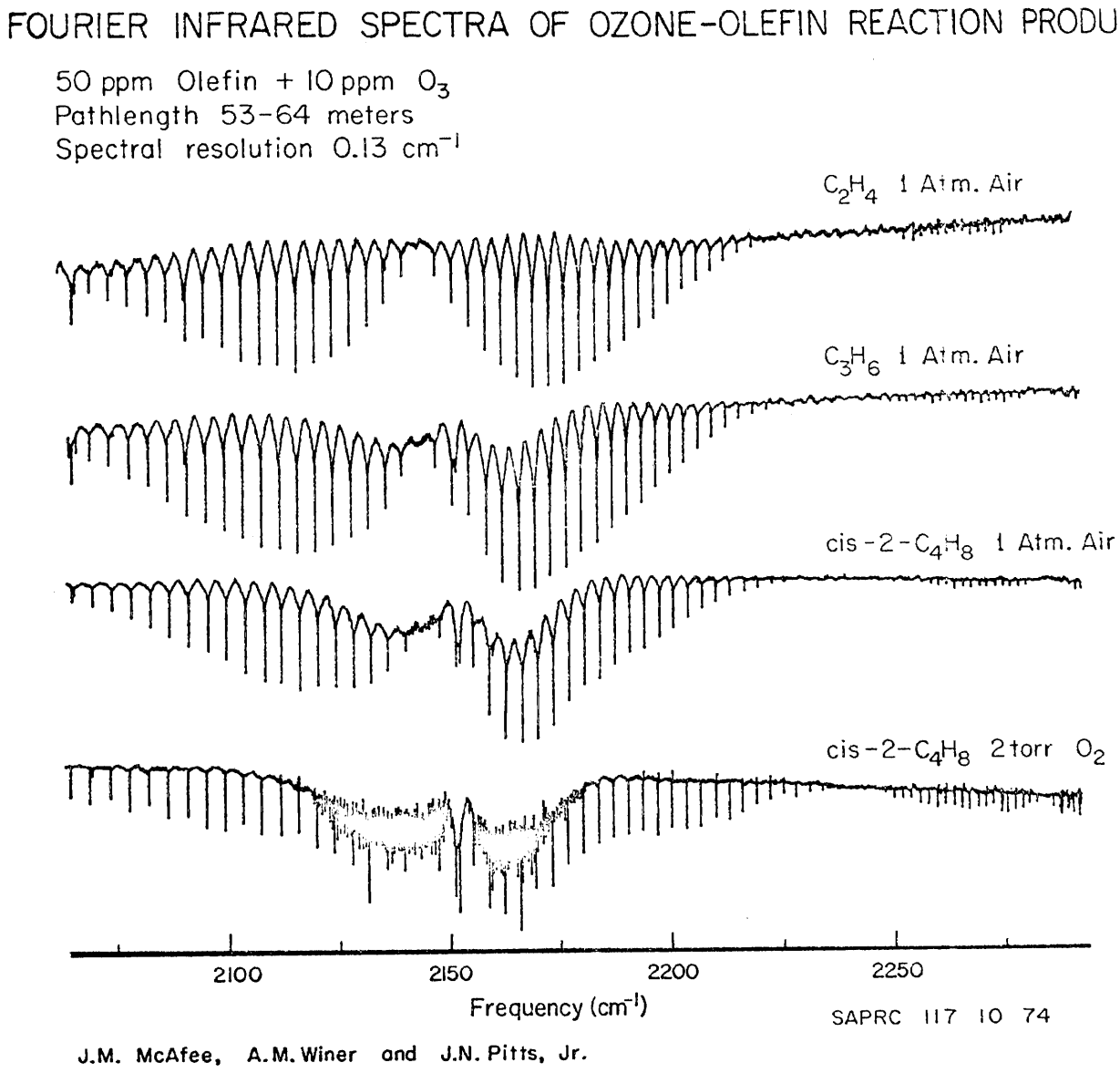

FOURIER INFRARED SPECTRA OF OZONE-OLEFIN REACTION PRODUCTS
50 ppm Olefin + 10 ppm O_3
Pathlength 53-64 meters
Spectral resolution 0.13 cm^{-1}
C_2H_4 1 Atm. Air
C_3H_6 1 Atm. Air
cis-2-C_4H_8 1 Atm. Air
cis-2-C_4H_8 2 torr O_2
2100
2150
2200
2250
Frequency (cm^{-1})
SAPRC 117 10 74
J.M. McAfee, A.M. Winer and J.N. Pitts, Jr.

Figure 10

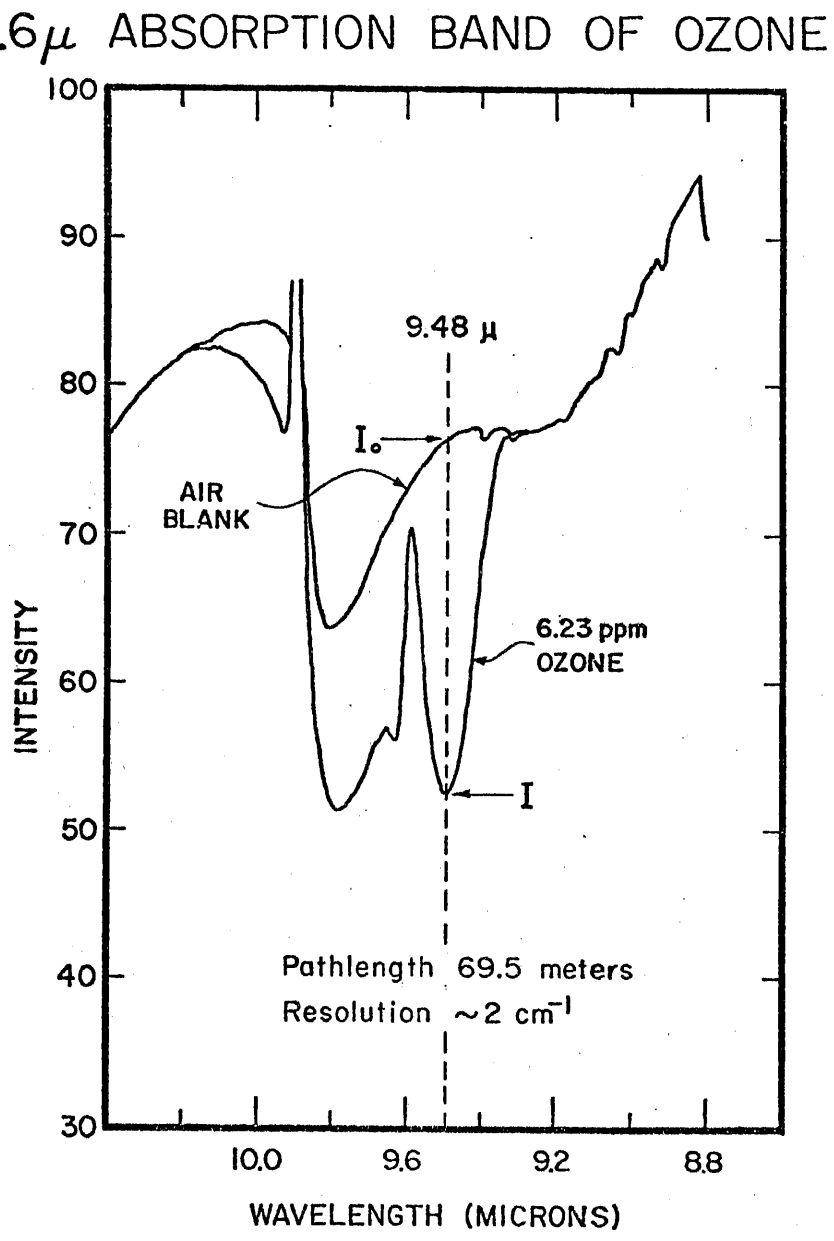


Figure 11. Ozone absorption band centered at 9.6 micron showing R-branch at 9.48 micron used to determine ozone concentrations.

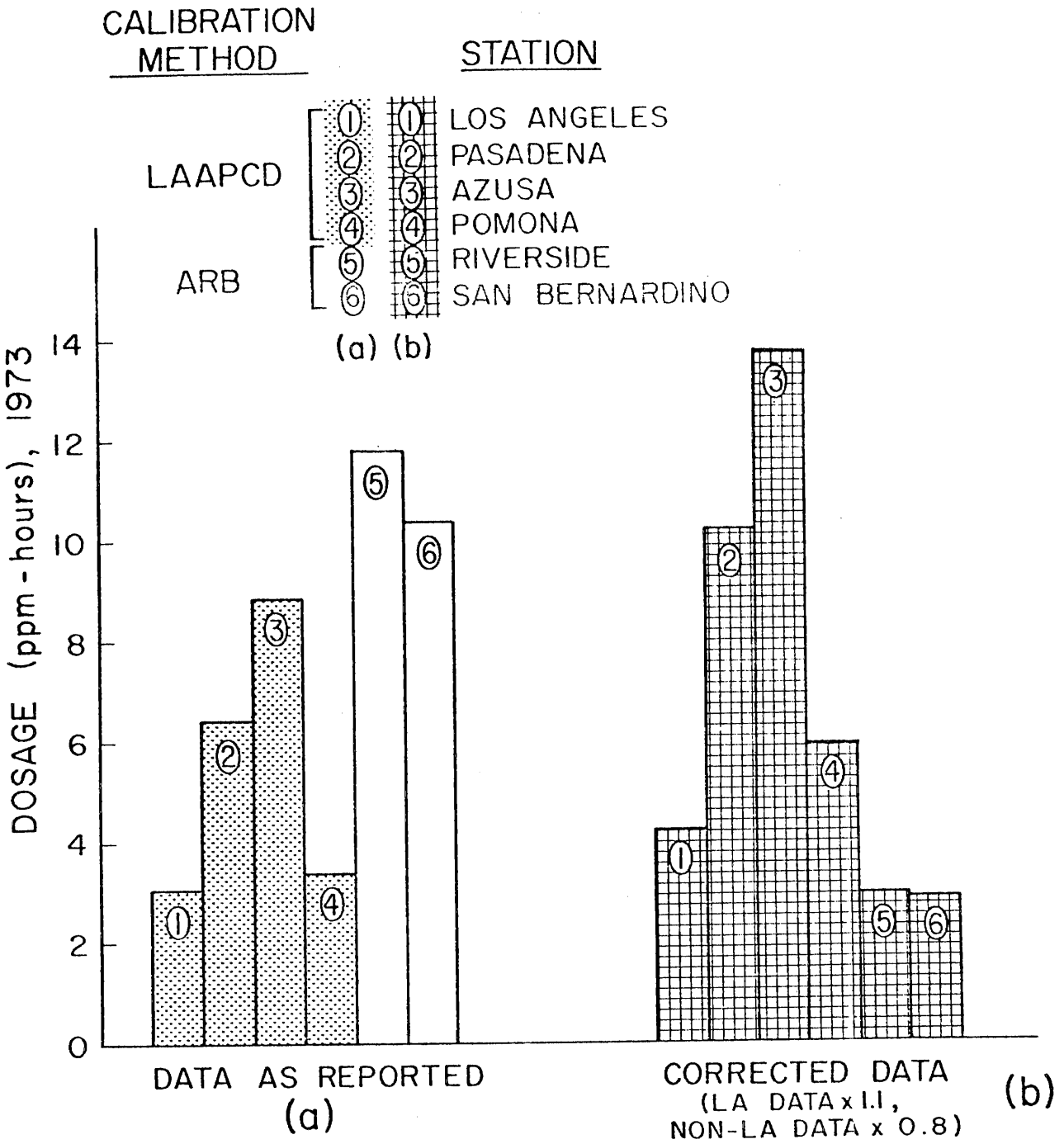


Figure 12. 1973 oxidant dosage greater than or equal to 0.20 ppm at six air monitoring stations in the South Coast Air Basin; a) data as reported; b) corrected data--LAAPCD data x 1.1, non-LAAPCD data x 0.8.

IMPLICATIONS FOR OXIDANT CONTROL STRATEGIES OF RESULTS OF SAPRC* HYDROCARBON-NO_x SURROGATE IRRADIATIONS

by

George J. Doyle
Associate Research Chemist
Statewide Air Pollution Research Center
University of California, Riverside

Data on ozone profiles resulting from six-hour irradiations of surrogate-NO_x mixtures in the SAPRC-ARB glass chamber were analyzed in a manner related to air quality standards (Figure 1). One result is a Dimitriades-style boundary curve separating the concentration region wherein ozone exceeds 0.1 ppm for 1 hour or more.

Two aspects of this diagram are noteworthy:

1. The extremely low nitrogen oxides required to avoid exceeding 0.1 ppm ozone for any hydrocarbon level exceeding 0.3 ppmC, confirming Dimitriades' earlier work, and
2. The finding that mixtures lower in hydrocarbon to NO_x than a ratio of 3.3 never exceed 0.1 ppm ozone in six hours.

The data were analyzed in terms of dosages accumulated during the 6-hr irradiations. These data were smoothed as illustrated in Figure 2. The functional form fitted was chosen as the simplest rational expression capable of reproducing the sharp peak at a critical nitrogen oxides concentration. A second example is also shown in Figure 3.

These and similar data at other hydrocarbon concentrations were melded to obtain a representation of the dependence of dosage on hydrocarbon and nitrogen oxides, jointly. The result, plotted as a contour diagram in logarithmic scales, is shown in Figure 4. Constant ratios on this sort of

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*Statewide Air Pollution Research Center

graph are 45° lines, some of which are shown in the figure. This diagram brings out the important fact that the ratio of hydrocarbon to nitrogen oxides is not the only important parameter; in some regions, the absolute concentration level also plays a large role, particularly along ratio lines crossing high dosage contours near the peak or ridge of this oxidant mountain. The federal standard is usually just violated along some contour above and near the 20 ppm-min contour.

In order to illustrate the implications of some recent air monitoring data, they may be compared to the dosage contour diagram of Figure 4. The air monitoring data are presented in Figure 5 in a smoothed form as a contour diagram of the joint distribution of observed nitrogen oxides and hydrocarbon concentrations in the morning hours (6-9 a.m.) at LAAPCD Los Angeles Downtown station during 1973. The solid lines represent probability contours. The hydrocarbon scale represents an internally consistent, although not necessarily accurate, estimate of reactive hydrocarbon concentration corresponding to the quoted total hydrocarbon and methane concentration data. In essence, the estimate resulted in calculating reactive hydrocarbon as total hydrocarbon less 1.4 divided by three. No attempt was made to estimate measurement error and correct for the associated variance. Two curves show the regression lines of the means and the modes. These curves approach a ratio near three at higher concentrations but show an apparent influence due to excess nitrogen oxides emissions not well correlated with hydrocarbon emissions at los concentrations (and, presumably, unstable atmospheric conditions).

Now, let us superimpose Figure 5 on Figure 4, as shown in Figure 6. This superimposition assumes that reactive hydrocarbon estimates are accurate. Note that initial conditions at this station seldom seem to be conducive to high oxidant dosages, only the 10^{-3} probability contour passing near the dosage peak. However, the 20 ppm-min dosage contour passes near the peak of the probability distribution, implying frequent violation of the state air quality standard for oxidant and, considerably less frequently, more severe episodes, in qualitative agreement with the facts. Of course, not every favorable initial condition can result in oxidant because of random atmospheric influences during transport.

Transport without further emissions in the South Coast Air Basin usually result in composition movement downward along the 45° line through the initial point, due to dilution. Addition of automotive exhaust emissions will cause a trend toward the ratio line characteristic of that source, presumably 3:1, according to this picture. Of course, the consequences of these dynamic processes are not predictable from these dosage curves, and are the subject of current research.

The implication of these diagrams for oxidant control is that the overlap of the two surfaces needs to be reduced. If these surfaces are at all accurately represented here, then control should jointly affect nitrogen oxides and reactive hydrocarbon in a ratio of about 1 to 3 or more (if nitrogen oxides are to be reduced). For moderate changes, this should have small effect on frequency of federal standard violation but larger downward effect on the frequency and severity of the high oxidant episodes.

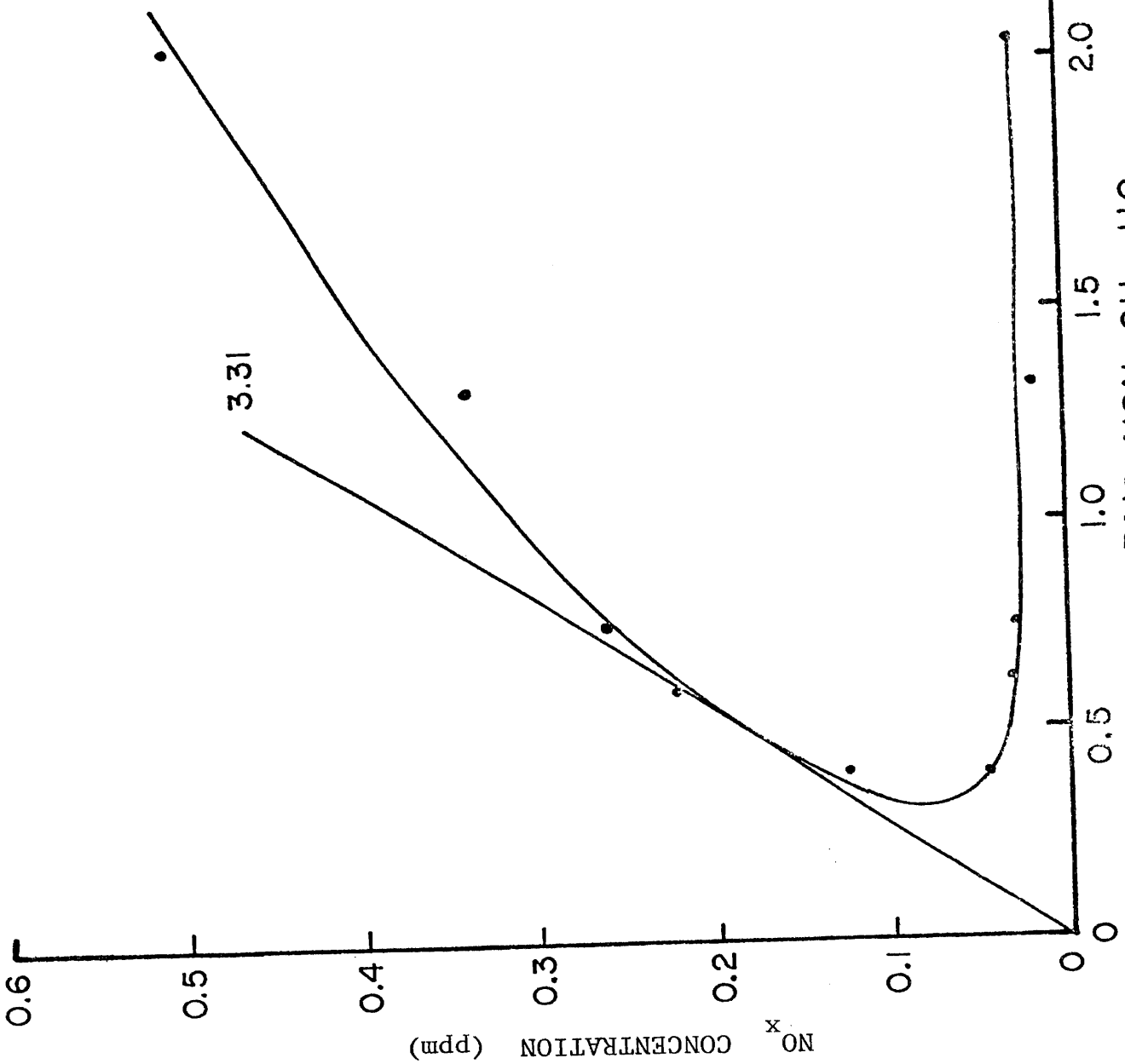


Figure 1. Estimate of Boundary Curve for 0.1 ppm Ozone (1 hr or less)

OZONE DOSAGES AND SMOOTHING FUNCTION FOR SAPRC SURROGATE IRRADIATIONS

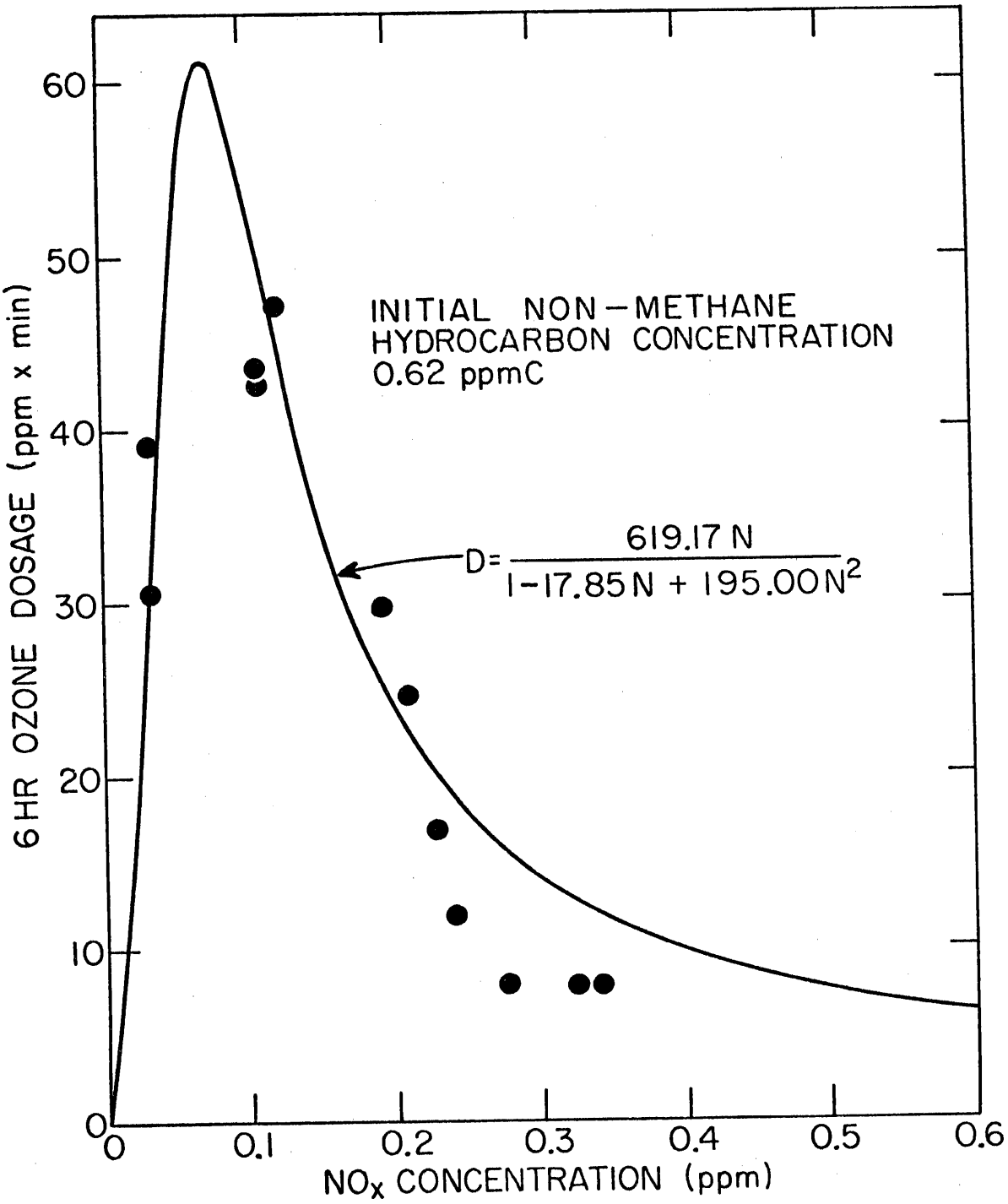


Figure 2

OZONE DOSAGES AND SMOOTHING FUNCTION FOR SAPRC SURROGATE IRRADIATIONS

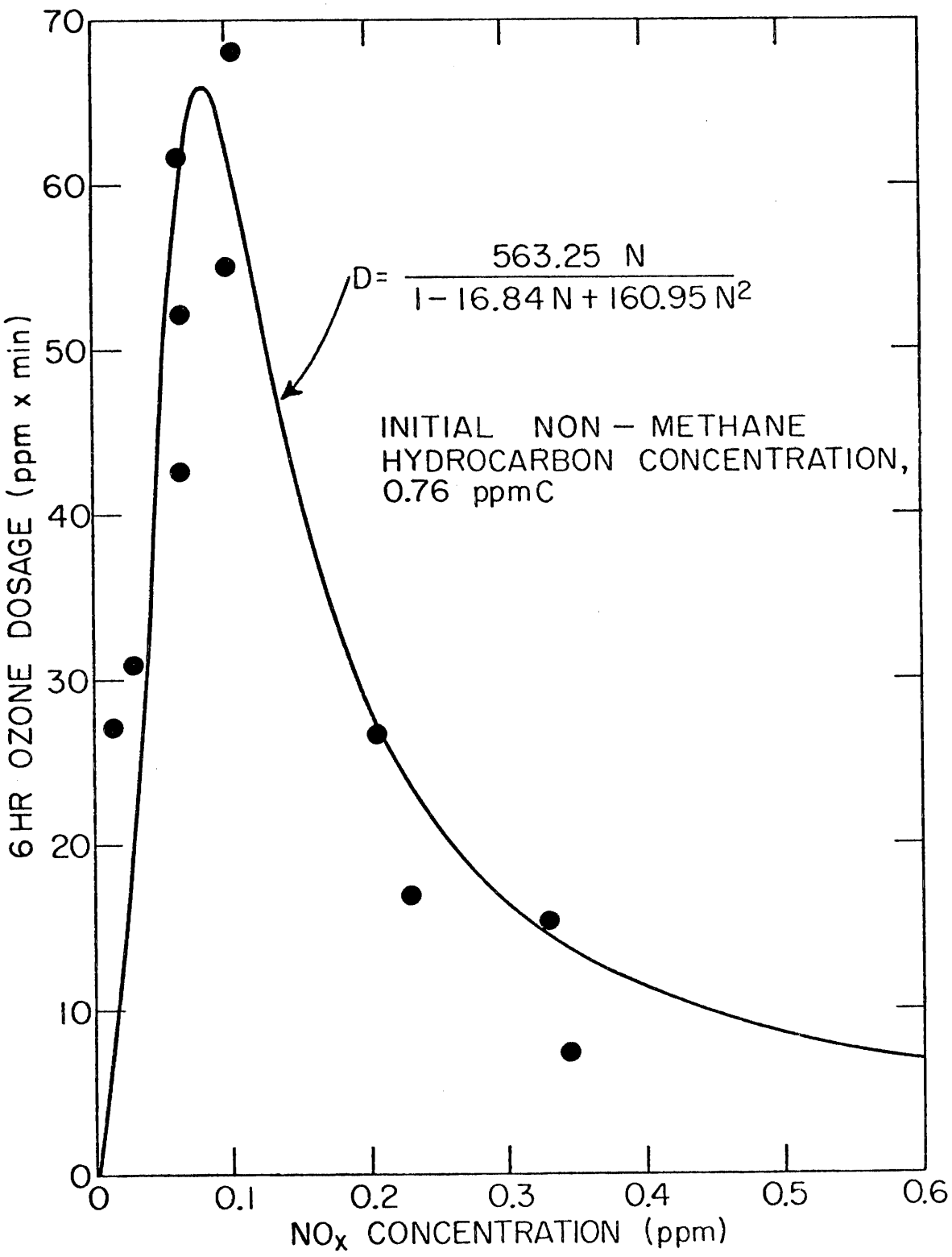


Figure 3

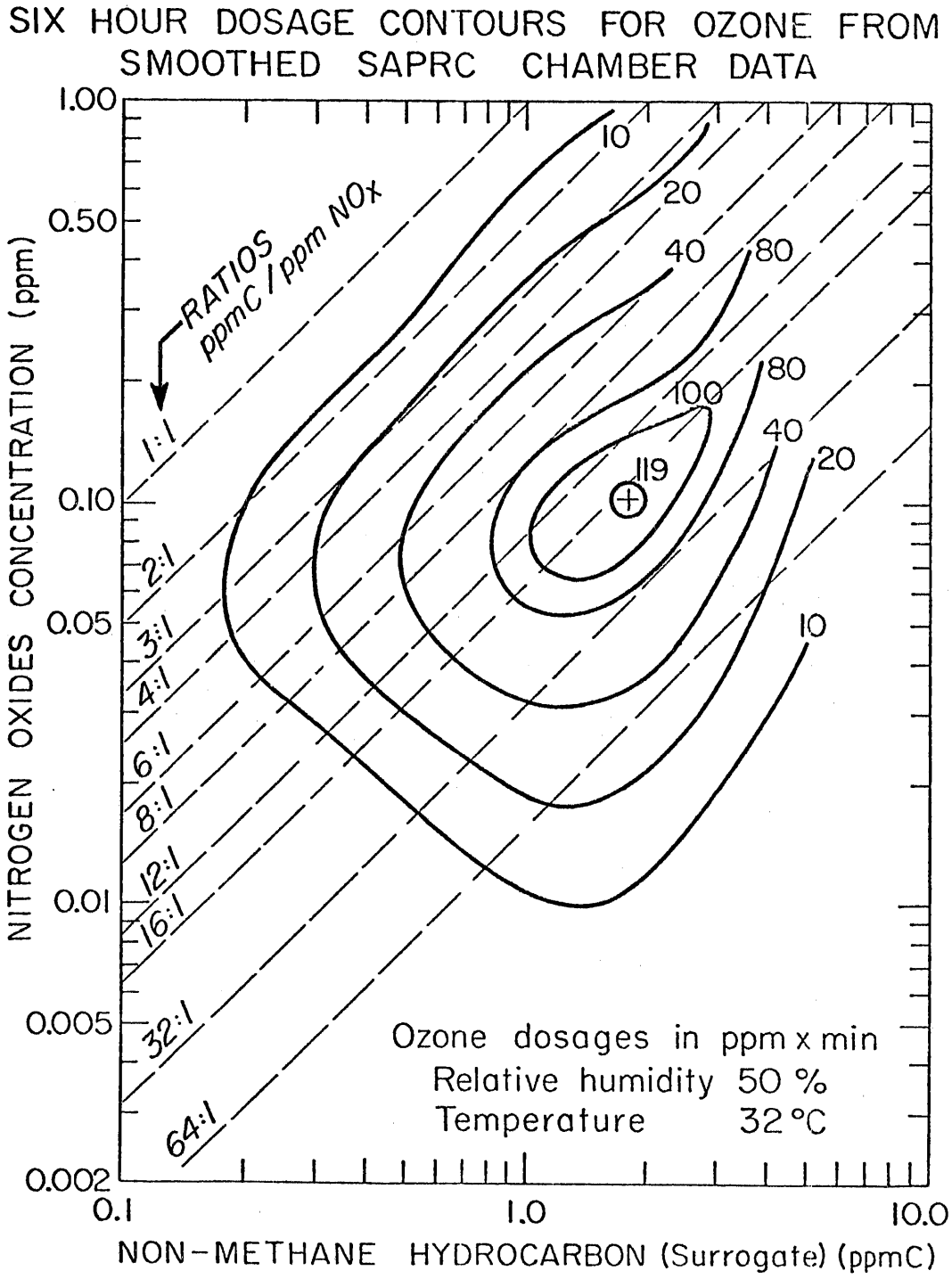
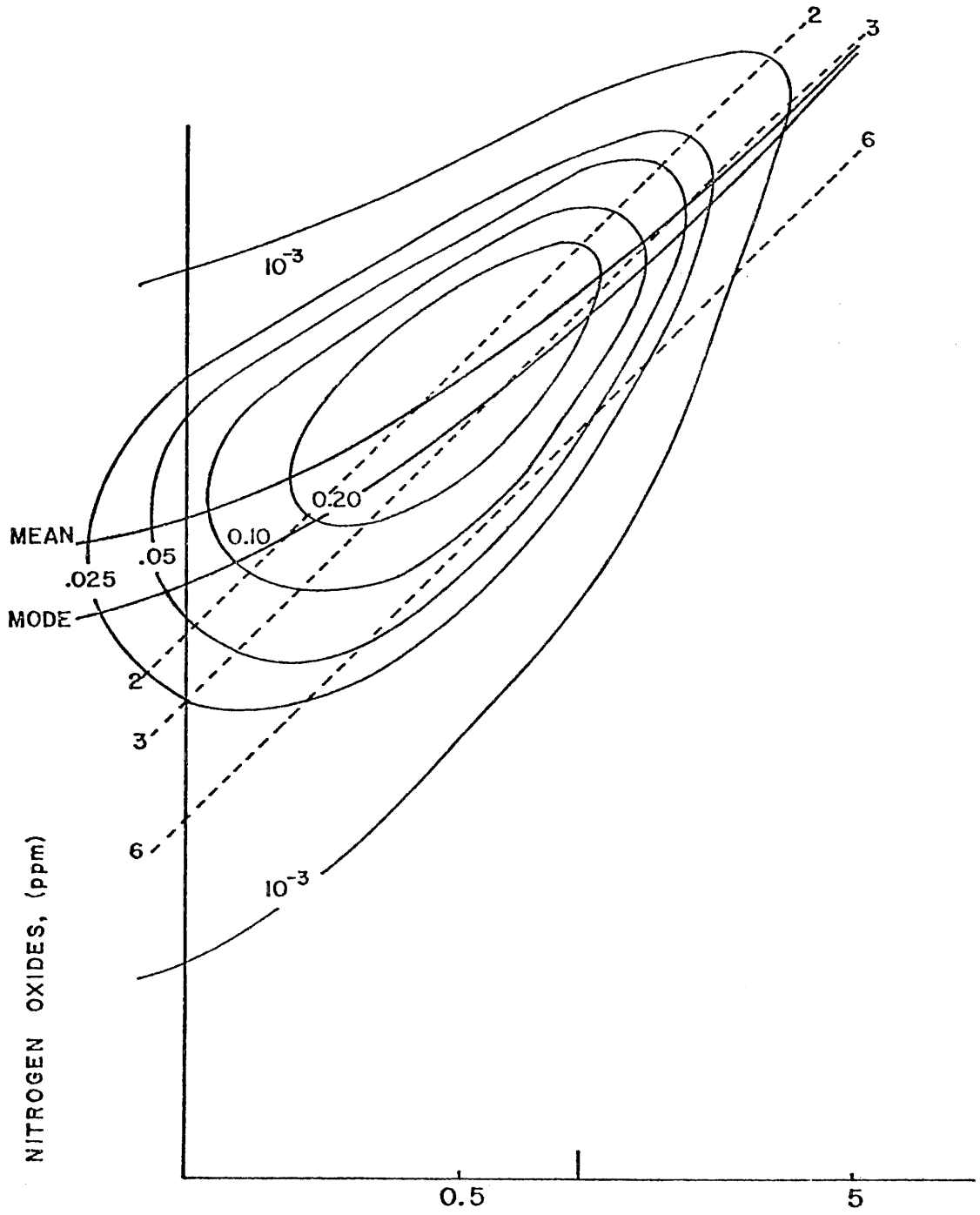

SIX HOUR DOSAGE CONTOURS FOR OZONE FROM SMOOTHED SAPRC CHAMBER DATA
NITROGEN OXIDES CONCENTRATION (ppm)
1.00
0.50
0.10
0.05
0.01
0.005
0.002
RATIOS ppmC / ppm NOx
1:1
2:1
3:1
4:1
6:1
8:1
12:1
16:1
32:1
64:1
10
20
40
80
100
80
40
20
10
119
Ozone dosages in ppm x min
Relative humidity 50 %
Temperature 32 °C
0.1
1.0
10.0
NON-METHANE HYDROCARBON (Surrogate) (ppmC)

Figure 4



SMOOTHED DATA LA APCD #1, 6-9 AM, 1973

Figure 5

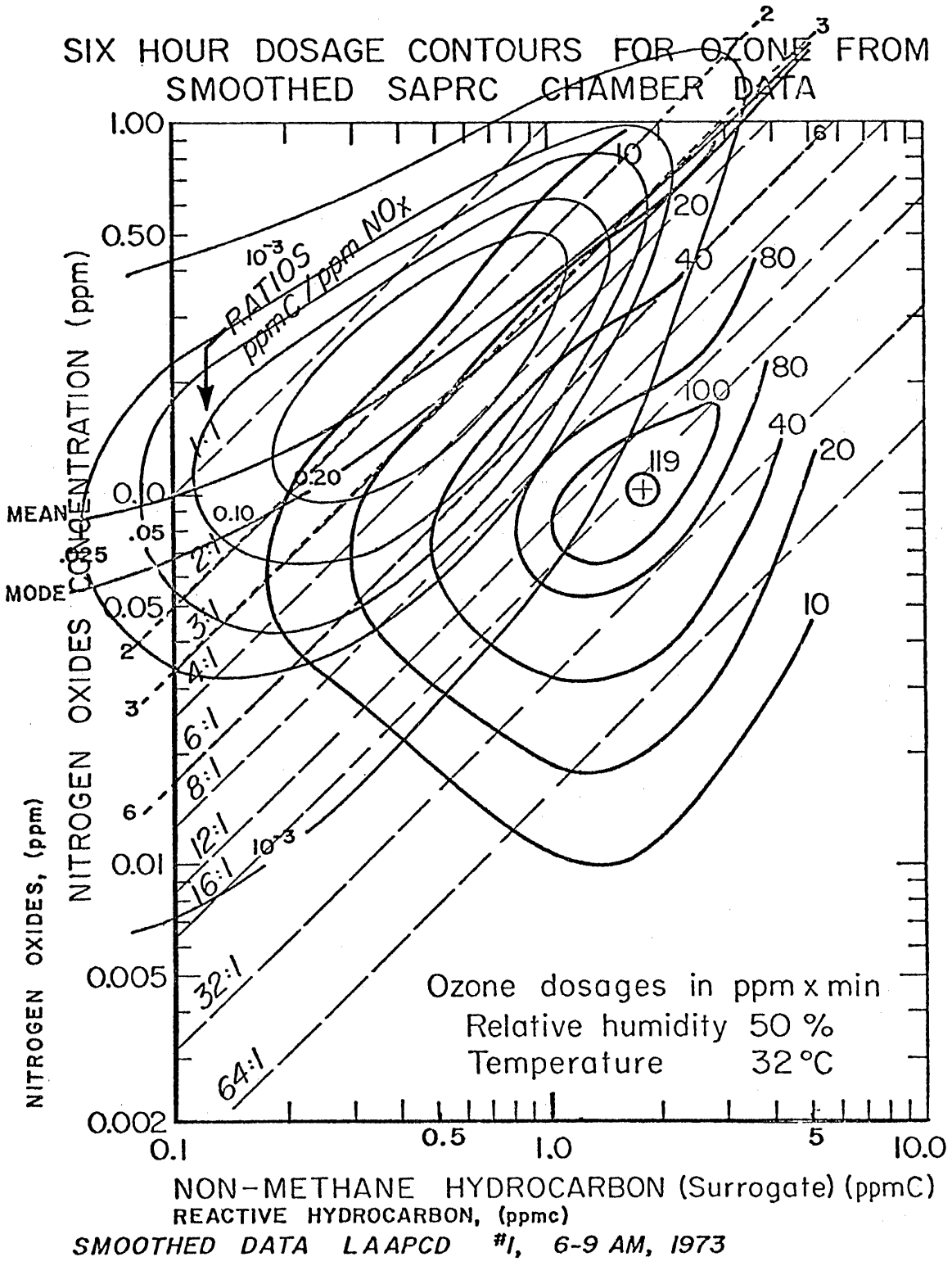


Figure 6. Comparison of 1973 air monitoring data with ozone dosage contour diagram by superimposing Figure 5 on Figure 4.

THE USE OF SMOG CHAMBER DATA IN FORMULATING OXIDANT CONTROL STRATEGIES

by

Basil Dimitriades
Chief, Atmospheric Reactivity Section
Chemistry & Physics Laboratory
Environmental Protection Agency
Research Triangle Park, North Carolina

In discussing the use of smog chamber data in formulating oxidant control strategies, I will attempt to make a case for the merits and drawbacks on such use of smog chamber data. Further, since the only alternative source of pertinent information is the aerometric data, I will attempt to discuss and judge merits or drawbacks of the smog chamber data in comparison with the merits and drawbacks of the other option, the aerometric data.

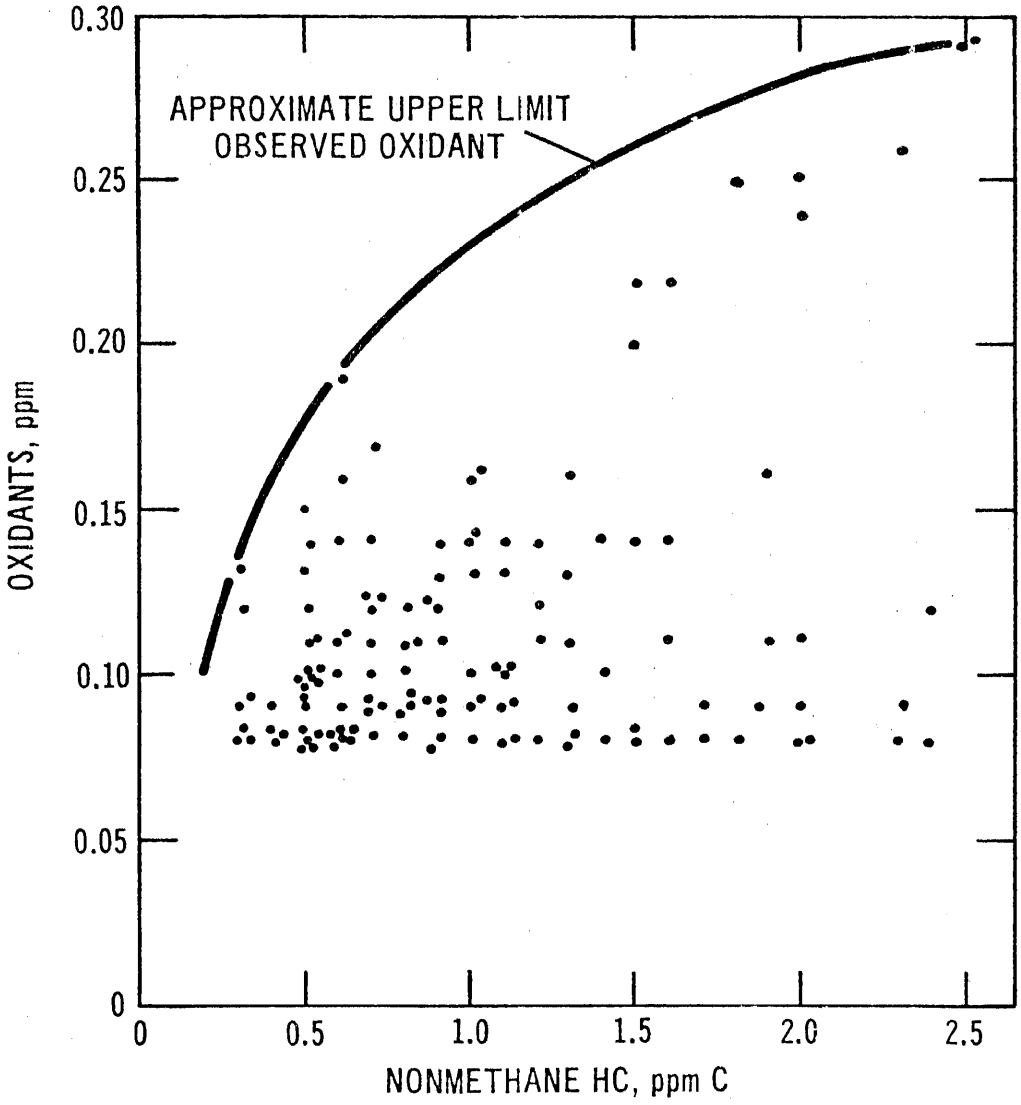
At the time when development of an oxidant control strategy was first considered, it was judged that such a strategy should be based preferably on aerometric data--smog chamber data should be the second choice. Such a judgment was at that time justified by 3 premises. The first premise was that any information on oxidant-precursor relationships, based on real atmosphere data, had inherent validity, which certainly is not the case with the smog chamber data. The second premise, which may not have been well appreciated, was that an oxidant control strategy based on aerometric data should be expected reasonably to be effective also in controlling other smog constituents that form along with oxidant. This is important because it constitutes a part of the rationale underlying the need for oxidant control. An oxidant control strategy based on smog chamber data alone may not necessarily be effective in controlling those other smog constituents, because such data usually pertained to oversimplified

simulations of the real atmosphere. Finally, the third premise in favor of the aerometric data was that smog chamber methodology and data, at that time, were lacking in several respects.

These premises and justification of the aerometric data, while acceptable a few years ago, are no longer valid. Recent developments have changed the picture considerably. Thus, after having examined the aerometric data taken in the CAMP stations for several years, investigators have come to realize that such aerometric data are much less interpretable than originally thought. Also, there is now a better understanding of and more confidence in the smog chamber data. These developments have changed the picture to the point that the question of the relative utility of aerometric data and smog chamber data should be reexamined. In the following discussion an attempt is made to provide such a reexamination.

It is submitted here that aerometric data such as that taken and used thus far to obtain empirical oxidant/hydrocarbon/nitrogen oxides (O_x/HC/NO_x) relationships--that is, the CAMP data--can have only suggestive value. It will be nearly impossible to delineate the precursor effects and the meteorological factor effects (upon oxidant) based on such aerometric data alone. This is because the emission rates and composition (HC/NO_x, HC composition) of the oxidant precursors vary very little relative to the variation of the meteorological factor. Therefore, relationships such as the well known "upper limit" curve (figure 1) do not necessarily depict the O_x-to-precursor dependency; rather, they depict the O_x

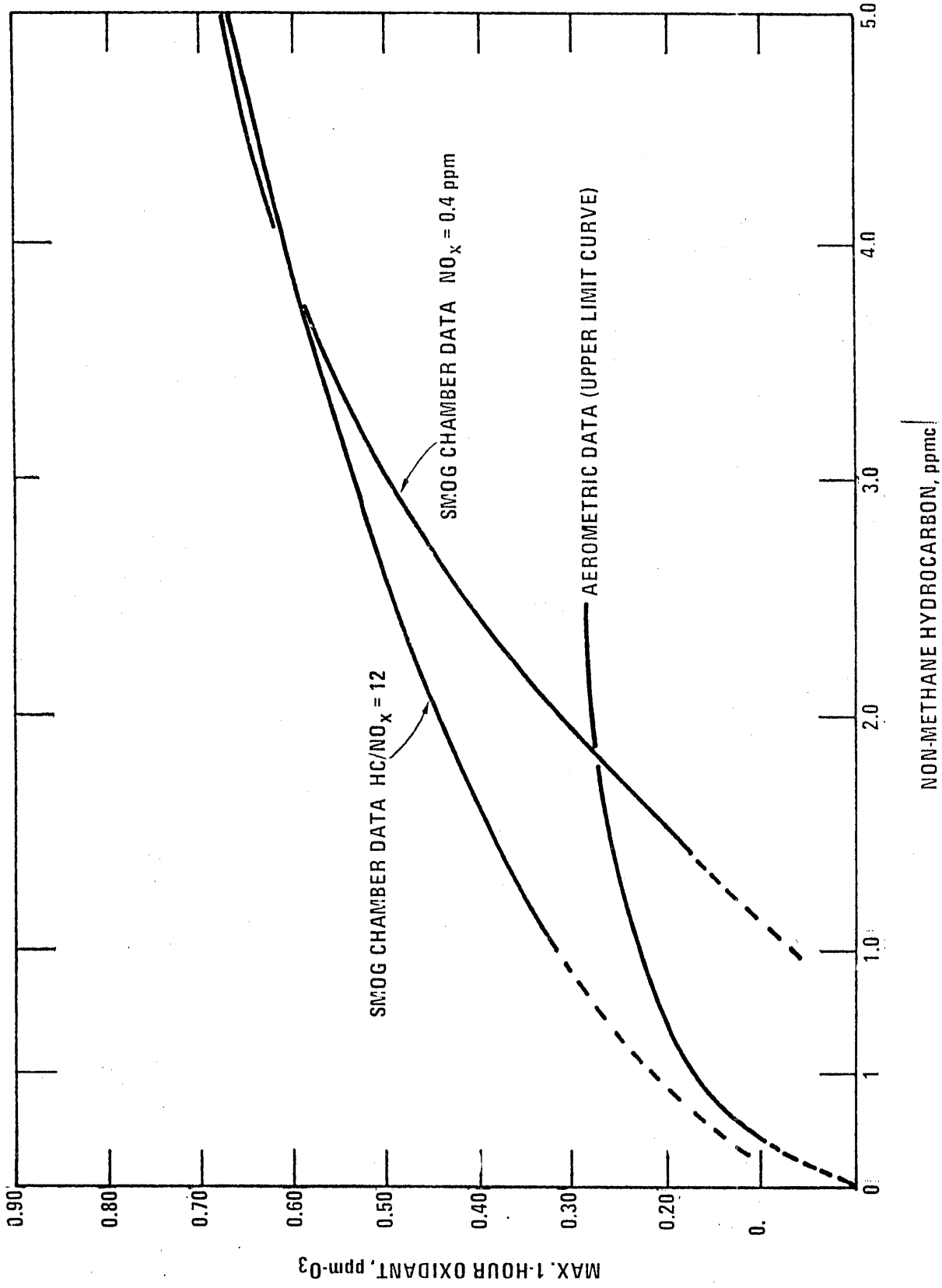
Figure 1. MAXIMUM DAILY 1-HOUR-AVERAGE OXIDANTS AS A FUNCTION OF 6-TO-9-A.M. AVERAGES OF NON-METHANE HYDROCARBON (CAMP DATA FROM FOUR USA CITIES)



dependence on meteorological factors. To further explain, the upper limit curve depicts the dependence of oxidant on degree of emission dispersion, since varying dispersion is what causes variation in HC concentration. It has been assumed that dispersion of all reactants and products has the same effect as reduction of the HC reactant alone insofar as oxidant reduction is concerned. However, this assumption is not necessarily correct as it is explained next.

To compare the effect of dilution with the effect of unilateral reduction of the HC reactant, it is necessary to use smog chamber data. Such data, taken from one study[1], are depicted in figure 2. These data show that while dispersion and HC control have similar qualitative effects on oxidant, their quantitative effects disagree to an extent depending on the NO_x concentration factor. Also, it should be noted here that these smog chamber data were taken using a chamber with fixed radiation intensity. Had the light intensity been varied to simulate natural sunlight, the extent of disagreement would be even greater, as some recent studies indicate. In summary then, the chamber data suggest that dispersion and HC control have similar effects qualitatively, but in quantitative terms, unilateral control of the HC reactant seems to have a much greater effect on ambient oxidant than dispersion. This leads to the conclusion that relationships constructed from aerometric data alone (such as the upper limit curves presently in use) can have severe inherent limitations with respect to deriving numerical control requirements.

Figure 2. Oxidant-Hydrocarbon Relationships from Smog Chamber and Aerometric Data.



Aside from these limitations, there is also the problem of the enormous quantity of data needed to construct an acceptable upper limit curve. The problem here is not one of logistics or expense only; rather it is the question whether it is feasible to gather sufficient data without introducing some bias. To obtain the large amount of data needed, one could either obtain and pool together data from several cities or use data taken in a single city but during a period of several years. In either case, the data would suffer from unavoidable biases introduced by the so-called "siting" factor, that is, the factor related to the position of the monitoring station relative to the position of the emission sources. Thus, in the single city case, the siting factor is likely to change in a period of several years because of some shift of emission sources in the vicinity of the monitoring station; data taken during these years will be internally inconsistent. Likewise, data from several cities will also be inconsistent because the "siting" factor cannot be the same (or have the same effect upon the air monitor measurements) from city to city. In conclusion then, "upper limit" curves not only have inherent limitations; they are also difficult to construct because it is not feasible to obtain consistent data in the amount needed.

The fundamental problem underlying the limited utility of the aerometric data to date is the inhomogeneity of the ambient atmosphere, especially in the lower layers where humans

and vegetation are. Non-uniform discharge of emissions into the atmosphere, their incomplete mixing with air, their varying reaction to form O_3, and the partial destruction of O_3 on surfaces, are factors that cause inhomogeneity to a degree such that the real atmosphere simply cannot be defined uniquely and at the same time meaningfully. It is mainly this problem of undefinability of the real atmosphere that led me to believe that the smog chamber data may have a certain advantage over the aerometric data and should be given further examination.

Smog chamber data have traditionally been distrusted for two main reasons. The first reason is that data obtained with different chambers showed considerable disagreement which we could not explain--the problem being not the disagreement itself, but the fact that this disagreement was unexplainable. The second reason is that there has never been evidence to show whether the smog chamber data are applicable to the real atmosphere. It is submitted here that these problems/questions still exist, but they no longer prohibit the use of smog chamber data; at least, there are things that can be done to alleviate these problems, as is explained next.

Table 1 shows the variation in some results among all chambers in use some 8 years ago[2]. It should be stressed here that the light intensity, light spectrum, and chamber design--in terms of surface-to-volume ratio (S/V) and material--varied extremely widely among the compared chambers. Thus, the light intensity factor k_d^1 varied from 0.2 to 0.4 min^{-1}, S/V varied

TABLE 1. RANGE AND AVERAGE VALUES OF PROPYLENE/NO_X REACTIVITY DATA OBTAINED WITH VARIOUS (9) SMOG CHAMBERS[2]

INITIAL CONCENTRATIONS OF PROPYLENE/NO_X (ppmC/ppm)	R_{NO_2}, ppb/min		MAX. OXIDANT, ppm	
	RANGE	AVG.	RANGE	AVG.
9/3	14 - 39	25.3	0.5 - 1.4	0.79
9/1.5	18 - 42	28.9	0.5 - 1.4	1.0
9/0.5	7 - 52	22.2	0.2 - 1.0	0.59
1.5/0.25	5 - 50	13.4	0.2 - 0.6	0.39

from 0.78 to 4.6 ft^{-1}, T varied by $\pm$ 15°F, and construction materials included Aluminum, stainless steel, Teflon, glass and even Nickel. Earlier studies and more recently studies conducted at Lockheed have shown that all these factors do affect smog chamber measurements and, therefore, the observed disagreement in results from those chambers is not necessarily a mystery[3]. Although it is not known yet whether the Lockheed findings are consistent with or can explain all of the (data) disagreement, the fact remains, nevertheless, that considering the differences in design factors among the chambers of the early studies, and considering also that these factors do affect chamber results, there is no reason to view this disagreement in chamber data as a mystery and, therefore, as a reason for distrusting the smog chamber method.

Chamber design factors are not the only factors affecting chamber results. Initial concentration and composition of the HC and NO_x reactants are also important factors. It is now known, from both experimental and modeling studies, that in smog chamber studies of the O_x/HC/NO_x relationships, the use of a single hydrocarbon reactant--instead of a hydrocarbon mixture--can lead to different conclusions. Furthermore, to simulate real atmosphere conditions in the chamber, it is imperative that an organic reactant mixture is used which is similar to that typically present in urban atmospheres, including different types of hydrocarbons as well as aldehydes, rather than a single hydrocarbon.

In conclusion then, there is no justification for regarding the disagreement among the various chambers as a problem invalidating the smog chamber method. Chambers of different design give different results; however, this is to be expected and it is now fairly well understood why the results differ.

The next question one may raise is: Of the various chambers and chamber conditions, which is the one that gives us the "correct" results? This question is the essence of the second problem or reason for distrusting the smog chamber data. The problem is that there is no evidence to show whether the data from a smog chamber are applicable to the real atmosphere. This is a real problem, but again, not an insurmountable one, as shown next.

The real question here is how to determine whether the $O_x/HC/NO_x$ relationships obtained with a smog chamber are the same as those in the real atmosphere. It is offered here that there are two conceivable answers or methods by which such a determination can be made: The "simulation" method, and the "direct" method. In the simulation method, it is assumed that the chamber results are valid, that is, the same as those in the real atmosphere, provided the important real atmosphere conditions are simulated closely in the chamber. In the direct method, the judgment concerning the validity of the smog chamber data is based on a direct comparison of pertinent smog chamber data with atmospheric data.

The success of the simulation method depends on how well one can define and how closely can simulate real atmosphere conditions in the smog chamber. The most important conditions are those related

to the following factors:

- -Intensity and spectrum of radiation, and diurnal variation of intensity and spectrum
- -Composition of organic reactant
- -Mixing of initial reactants with diluent air and with fresh emissions
- -Type and condition of surface, and surface-to-volume ratio
- -Magnitude and diurnal variation of temperature (T) and relative humidity (RH)

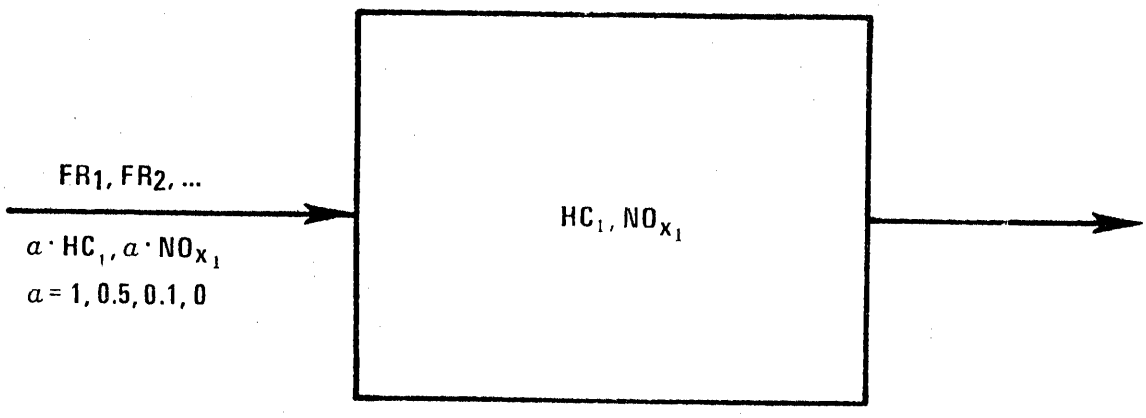
Of these, only the radiation factor is uniquely defined; the ambient condition in this case can be virtually duplicated by using an outdoor smog chamber. The T and RH factors are also relatively easy to duplicate. The organic reactant composition factor also can be simulated closely by using automotive exhause mixed with gasoline and other known vapors, or a carefully designed synthetic mixture. This leaves the "mixing" factor and the "surface" factor.

The mixing factor is difficult to simulate only because atmospheric mixing cannot be defined uniquely. This would be of no concern if the mixing factor had no strong effect on oxidant. However, in the lack of relevant evidence, one must assume that the mixing factor does influence oxidant formation, and that the rational way of uniquely defining this factor is by defining the mixing conditions that cause the highest oxidant concentration. Thus, to obtain the oxidant yield corresponding to a given combination of reactants, e.g. HC_1, NO_{X_1}, a series of smog chamber tests should be conducted, in which dilution and fresh reactant

injection would be varied as shown in figure 3, and the highest oxidant-yield result should be taken as the applicable result. This should be an adequate solution of the problem of simulating atmospheric mixing in the chamber.

The atmospheric condition related to the surface factor is perhaps the most difficult one to simulate in the smog chamber with confidence. Laboratory data, namely, the evidence regarding the existence of "virgin surface" and of S/V effects indicates that the surface factor may be an important one. Photochemical models also predict an important role for the surface factor. All these indications, however, are based on results from experimental systems in which the S/V (1.3 - 2.7 ft^{-1}) is much higher than in the real atmosphere. For example, comparison of a 100-cu. ft chamber with smog laden ambient air shows the chamber S/V to be at least 3 orders of magnitude greater than the S/V in the real atmosphere. Although the ground surface (buildings, trees, etc.) was not included in the calculation of the real atmosphere S/V, the chances are that the S/V in the chambers that manifested the surface effects is still greater--perhaps considerably--than the S/V in the real atmosphere. This means that to better simulate (in the smog chamber) the real atmosphere surface conditions, one should use a chamber in which the surface effects are minimized. Based on the Lockheed findings, the chamber should be made of Teflon and be as large as practical, e.g. 1000 cu. ft (S/V = 0.6 ft^{-1}).

Figure 3. Depiction of Smog Chamber Operated Under Varying Dynamic Conditions.



In summary then, the "simulation" method for obtaining valid smog chamber data, requires:

-Use of a Teflon film chamber of 1000-cu. ft or so capacity

-Operation outdoors, preferably within a smoggy area (where sunlight intensity is affected by smog)

-Irradiation of given reactant mixture under those conditions, dynamic or static, that yield the highest O_3 concentration

The direct method, in actuality, is an extension or refinement of the simulation method. In the direct method, data are obtained as prescribed by the simulation method, and are then compared with atmospheric data for the purpose of either validating or modifying the simulation method. The question to be asked here is what atmospheric data are needed for this purpose and whether it is feasible to obtain such data.

Ideally, one would like to obtain aerometric data on HC and NO_x and on the O_3 that formed from such HC and NO_x--such data would be directly comparable with the smog chamber data. This type of aerometric data, however, does not exist. There is an abundance of aerometric data that relate O_3, HC, and NO_x empirically. However, the empirical relationship between O_3, HC, and NO_x is not of interest here; what is of interest is the cause-effect relationship because this is what is obtained from the smog chamber data.

The LARPP study[4] is an effort intended to provide, among other things, aerometric data on the cause-effect relationship between O_3 and the O_3-precursors. However, whether this effort will be successful in this respect is uncertain at this time, for the following reason. The LARPP study was designed so as to provide

the requisite data on HC, NO_x, O_3, dilution, and fresh reactant injection, in a parcel of urban air as such air is moving downwind. The usefulness of such data depends to a large extent on the degree of homogeneity within the air parcel. Although the LARPP data have not been analyzed to conclusion yet, there are indications that a rather high degree of inhomogeneity might exist. Nevertheless, the LARPP data, conceptually, at least, illustrate the type of data needed to validate or develop the direct smog chamber method. Specifically, the data needed--to be obtained (with time) in a moving parcel of urban air--as follows:

-HC_t, HC_{GC}, aldehydes, NO, NO_2, O_3, PAN

-Dilution as determined by use of a tracer

-Total light and UV light

-T, RH

Such data would be used to (a) construct a time profile of the smog forming process as such process occurs in the real atmosphere, and (b) to duplicate the reaction system in the smog chamber and obtain a time profile of the same process as it occurs in the chamber. If the two time profiles, based on several tests, are reasonably similar, this would constitute an adequate validation of the chamber system. If the two time profiles are drastically different, then additional research would have to be done to further develop the chamber method.

In summary then, the viewpoint submitted here regarding the development and use of a valid smog chamber system is as follows. The simulation method, as described here, would give acceptable data.

For more confidence, the smog chamber data should be validated through with the aerometric data. For this latter purpose, the LARPP data could be used in a first try. If the LARPP data turn out to be inappropriate or inconclusive, then field studies should be conducted, specially designed to meet the objective of developing a valid smog chamber methodology.

Assuming next that an acceptable smog chamber system exists and that its data are accepted to be applicable to the real atmosphere, the question still remains of how can these chamber data be used to calculate control requirements for a given region. The answer proposed here is illustrated in figure 4 which shows what might be the $O_x/HC/NO_x$ diagrams derived from "acceptable" smog chamber data. The derivation procedure consists of the following steps:

1. From the value of the highest O_3 concentration measured in the region under consideration during the reference year, identify the specific O_x isopleth applicable to the region.

2. Determine the HC-to-NO_x ratio for the region. Such a determination is relatively easy--certainly much easier than the determination of the max HC and max NO_x--and can be estimated from aerometric data as well as from emissions inventory data.

3. From this HC/NO_x ratio value, define the O_3 isopleth point that denotes the HC and NO_x concentrations that resulted in the highest measured O_3 concentration.

4. Calculate the percent reduction of NO_x that is required in order to meet the air quality standard for NO_2. This and allowances for unavoidable variation of NO_x downward define the lowest NO_x concentration expected to occur in the future.

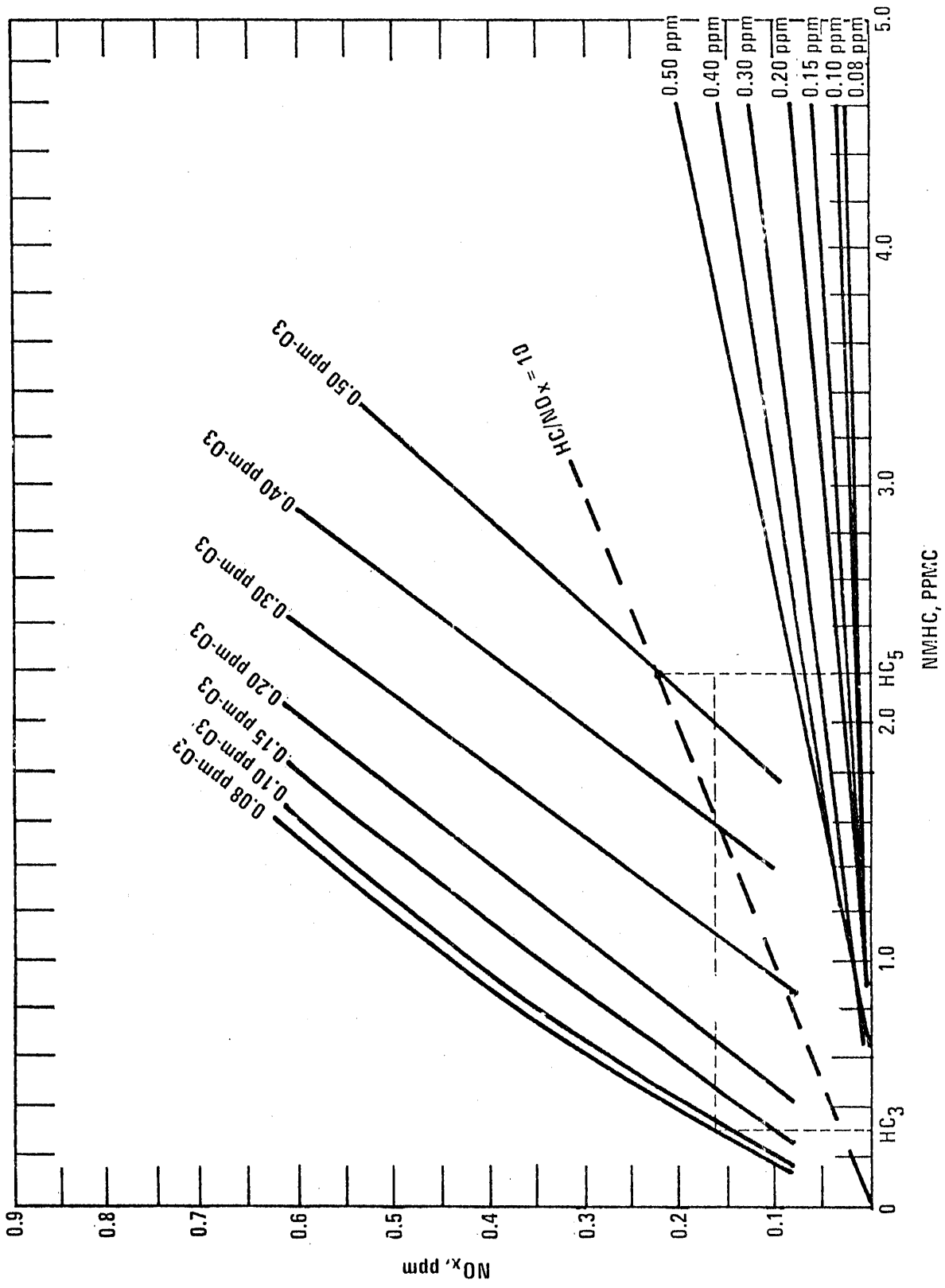


Figure 4. EQUAL RESPONSE LINES REPRESENTING COMBINATIONS OF NMHC, NO_x CORRESPONDING TO FIXED O_3 LEVELS

5. Using the NO_x value established in step 4 and the diagrams of figure 4, define the HC level corresponding to 0.08 ppm O_3.

6. From the HC values established in steps 3 and 5, calculate the degree of HC control required in order to achieve the O_3 standard.

It should be mentioned that another method has been proposed for using smog chamber data to develop oxidant control strategies. This other method is the one advocated by the Los Angeles County (LAAPCD) investigators and described recently in some detail.[5] The LAAPCD method, briefly, is based on a combined use of smog chamber and aerometric data and has a totally empirical nature. Specifically, $O_x/HC/NO_x$ relationships were derived from a LAAPCD smog chamber study; the smog chamber oxidant values were then correlated with observed maximum oxidant concentration values for the Los Angeles basin, and this correlation resulted in a relationship between ambient maximum O_3 and ambient max. HC and max NO_x.

A detailed critique of the LAAPCD method is outside the scope of this discussion. Nevertheless, this investigator wishes to state for the record that he consideres this method unacceptable mainly for the following reasons. Contrary to the claims implicitly made, the method is only an associative one; that is, the derived correlation equation between smog chamber oxidant and amibent oxidant defines an empirical relationship, and not a cause-effect relationship. As such, the correlation equation must be based on a large number of correlation data, if it is to be used with confidence. There requirements, however, were not met, and, what is even worse, they cannot possibly be met, ever. The available smog chamber oxidant

data and ambient oxidant data show no correlation whatever. The authors of the method selected a few (6) of these correlation data to derive their correlation equation; however, such selection was arbitrary. For such a method to be valid, a large number of correlation points should be used. However, since each point represents one year, it follows that the data needed must be obtained during a period of several years, and, as pointed out earlier, it is not feasible to obtain consistent atmospheric data during such a long period of time.

In summary, it is submitted that recent developments dictate that the bases of the current oxidant control strategies be reconsidered. With a reasonable research and development effort it would be possible to develop a smog chamber method that would provide bases for a more defensible oxidant control strategy than the one now in use. Development of valid smog chamber techniques is imperative also because such techniques may be the only one appropriate for studying photochemical pollution problems other than the oxidant problem.

REFERENCES

1. Dimitriades, Basil. Effects of Hydrocarbon and Nitrogen Oxides on Photochemical Smog Formation. Envir. Sci. & Technology, Vol. 6, No. 3, pp. 253-260, March 1972 (Note: Data were taken from this publication to construct diagrams in Figure 2.)

2. Smog Chamber Survey conducted by the Coordinating Research Council, Group CRC-CAP1-6. May 1971. Coordinating Research Council, 30 Rockefeller Plaza, NY, 10020.

3. Jaffe, R. J., F. C. Smith, and K. W. Last. Study of Factors Affecting Reactions in Environmental Chambers, Final Report on Phase III, from Lockheed Missiles and Space Co., Inc., to Coordinating Research Council, 30 Rockefeller Plaza, NY, 10020, and to U.S. Environmental Protection Agency, NERC-RTP, Research Triangle Park, NC, 27711, Sept. 1974.

4. Coordinating Research Council, Los Angeles Reactive Pollutants Project, CRC-APRAC-CAPA-12, 1974, Coordinating Research Council, 30 Rockefeller Plaza, NY, 10020.

5. B. Dimitriades. Proceedings of the Smog Chamber Conference, U.S. Environmental Protection Agency, Internal Report in press, 1975, U.S. EPA, NERC-RTP, Research Triangle Park, NC, 27711.

THE USE OF AMBIENT AIR DATA TO DEVELOP CONTROL STRATEGIES

by

A. Paul Altshuller
Director, Chemistry & Physics Laboratory
Environmental Protection Agency
Research Triangle Park, North Carolina

In this presentation I will take the view that each of the procedures, aerometric data analysis, smog chamber measurements, and kinetic models, have something to offer. I want to make some comparisons of the predictions that can be made by each of these procedures. It is my position that we need concurrent continuing work on all three approaches with periodic inter-comparisons. This is precisely the viewpoint the National Academy of Sciences panel report took with respect to concurrent utilization of these approaches to improve understanding of the relationships between atmospheric oxidant or ozone concentrations to hydrocarbons and NO_x. I do not propose to discuss another very important subject--long-range transport of pollutants. This is an important topic. We will be within the next year in a much better position to look into this phenomena based on results from the LAARP study out here, the ARB Tracer Studies, and a study which was run in the Midwest, referred to as the Midwest oxidant study. We have smog chamber studies in progress where we are simulating transport. I presume that as we move along with these experimental results, the modelers will also be attempting to simulate long-range transport. So that I would hope that a year from now we will be in a position to discuss meaningfully the quantitative aspects, at least in a preliminary fashion of long-range transport.

The first figure is the well-known upper limit curve which was constructed a number of years ago and placed in the nitrogen oxides criteria document.[1] *Plotting on the 6-9 AM non-methane hydrocarbon concentration versus maximum hourly maximal oxidants. This curve was based on the data available in the period between 1966 and 1968 for several cities, but the Los Angeles data primarily defined the upper portion of the curve while the Washington, D. C. and Philadelphia data define the lower portion of the curve. One of the types of problems with this curve cited in recent years is the fact that all the data from several cities was lumped together. If instead, each cities data was treated separately what would the nature of the results?*

Schuck and Papetti,[2] had oxidant data available in downtown Los Angeles, Azusa, and Riverside to develop the separate upper limit curves (Figure 2). For the same nonmethane hydrocarbon concentration a significantly higher oxidant concentration occurs at the downwind site in Azusa than we have nearer the source, DOLA. For other locations we do not have records for upwind-downwind sites. So only single site comparisons can be made in other cities. The CAMP data from Washington, D. C. assembled from between 1966 and 1973 is shown in Figure 3. The upper limit curve initially goes up steeply and then the slope decreases up to just under 0.25 ppm. Only a few days of data are available to define the upper portion of the curve. The Philadelphia CAMP data is shown in Figure 4. The points available define a straight line relationship. In terms of density of data the situation is better for both curves between 0.08 and 0.15 than above that concentration level. The Denver data, CAMP (Figure 5) is shown for approximately the same period of time. As for the curve constructed from Washington, D. C. data the lower portion of the curve is steep. Each of these three curves have different shapes and their intercepts at 0.08 ppm oxidant are somewhat different. These intercepts for Washington, D. C., Philadelphia and Denver are 0.1, 0.25 and 0.35 ppm carbon. Therefore, the curves and intercepts do appear different for these various sites within the limitations in available data.

Isopleth curves have been constructed[1] (Figure 6) relating maximum hr oxidant to hydrocarbon and nitrogen oxide concentration using Philadelphia, Washington, D. C., and Denver results from CAMP sites has been frequently done with smog chamber data. Here we have it for aerometric data. I suggested to Dr. Dodge of our laboratory that we take the Hecht-Seinfeld Dodge photochemical model and fit it to this sort of a profile. In doing this a hydrocarbon mixture represented by propylene and n-butane-nitrogen oxide was used because modelling of this system where we had good agreement with smog chamber experiments. A ratio of n-butane to propylene of 5 to 1 was used to roughtly simulate the paraffinic to olefinic ratio in the atmosphere. We assumed that the initial NO_2 concentration was 10% of the initial NO_x. The simulations were carried out for a 10-hour period at constant light intensity, K_a equals .3 min^{-1}. The model was fit to only one point on one isopleth. A single dilution factor of 5 x 10^4 min^{-1} was selected to fit that point. Comparison of the isopleths from the model with the previous experimentally

generated isopleths shows excellent agreement in the region at or below 0.6 ppmC (Figure 7). The model predicts that there will be no change in ozone formation above a hydrocarbon concentration of about .6 ppmC while the isopleths from the aerometric data have some curvature. There are no obvious additional reaction steps which will cause the model to predict the curvature observed experimentally. It isn't clear whether there is something wrong with the aerometric data or something wrong with that model in the region above 0.6 ppmC. The model can be used to generate isopleths over a wider region or more closely spaced than the experimental results. The paraffin to olefin ratio was adjusted to 3 to 1, 5 to 1, and 7 to 1, to simulate realistic ranging in this ratio. We found that there was extremely little change in the ozone maximum for the 10-hour simulation. The times to maximum vary substantially. And on the average the maximum 7 to 1 mixture occurred 2 hours later than the 3 to 1 mixture. However, this variation also did not result in any curvature in the higher hydrocarbon range. Therefore, changing the mixture ratio does not account for the difference in this region between these curves and the aerometric curves. Comparisons have been made of various initial hydrocarbon and nitrogen oxide concentrations of the oxidant concentrations from the model results, the smog chamber results (Dimitriades work)[3] and the upper limit curves (Table I). The agreement between model and smog chamber results in some cases is good but at other points of comparison the agreement is mediocre, for example, at 1 ppm carbon and 0.2 ppm of NO_x the smog chamber result predicts 0.22 ppm oxidant the model 0.21 ppm oxidant while at 0.75 ppm and 0.3 ppm NO_x the smog chamger results predict 0.08 ppm the model 0.14 ppm oxidant. In most cases the agreement is reasonable. This is so even when making comparisons in the hydrocarbon concentration above the 0.6 ppmC level. It should be noted that the dilution factor used in the model is not used for the smog chamber results. The smog chamber mixture was diluted automobile exhaust rather than the simple mixture used in the model.

A comparison also is given of the readings from the upper limit curves for Philadelphia, Washington and Denver at the same initial concentrations. The values off the Philadelphia upper limit curve are higher than from the model or smog chamber. The values from the Denver and Washington, D. C. upper limit curves fall into the predicted range of values. Since the model and the smog chamber predictions are both sensitive to hydrocarbon to NO_x ratios the comparison does indicate the range of variability which should be considered.

References Cited.

1. Environmental Protection Agency, Air Quality Criteria for Nitrogen Oxides, January 1971, Publ. No. AP-84.

2. Schuck, E. A. and Papetti, R. A., "Examination of the Photochemical Air Pollution Problem in the Southern California Area", Unpublished report.

3. Dimitraiades, B. "On the Function of Hydrocarbon and Nitrogen Oxides in Photochemical Smog Formation. U. S. Bureau of Mines Report of Investigations RI-7433, Sept. 1970.

Table I. Comparison of Predicted Oxidant Concentrations

HC, Cppm	NO_x, ppm	Photochemical Model	Smog Chamber Results	Upper Limit Curves Denver	Phila.	Wash.,D.C.
1.0	0.3	0.21	.13	0.145	(0.33)[a]	0.19
1.0	.25	.21	.15			
1.0	.2	.21	.22			
1.0	.15	.20	.24			
1.0	.10	.20	.27			
1.0	.05	.17	ND			
1.0	.025	.12	ND			
0.75	0.3	.14	.08	.14	.25	.17
.75	.25	.17	.11			
.75	.2	.18	.15			
.75	.15	.18	.18			
.75	.10	.17	.22			
.75	.05	.16	ND			
.75	.025	.14	ND			

[a] linear extrapolation

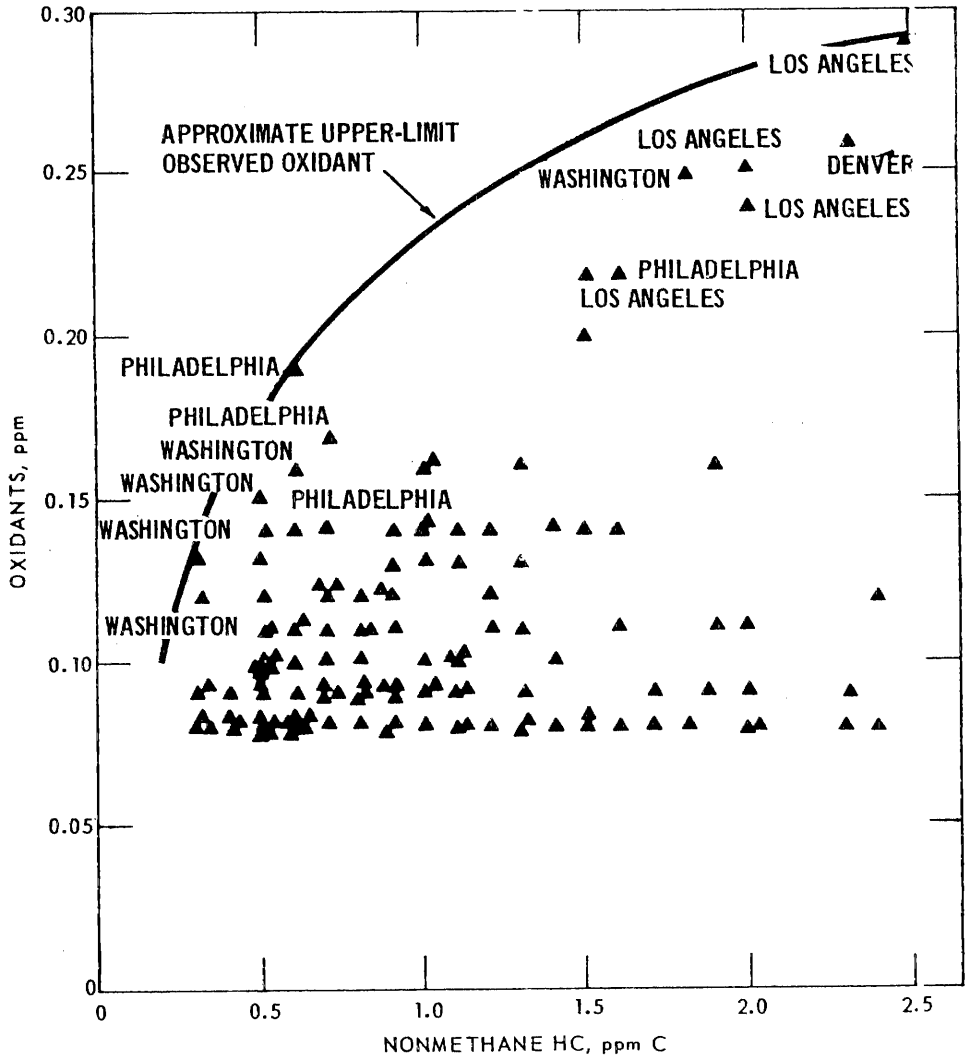


Figure 1. Maximum daily 1-hour-average oxidants as a function of 6-to 9-a.m. averages of nonmethane hydrocarbons at CAMP stations, June through September, 1966 through 1968, Los Angeles, May through October 1967.

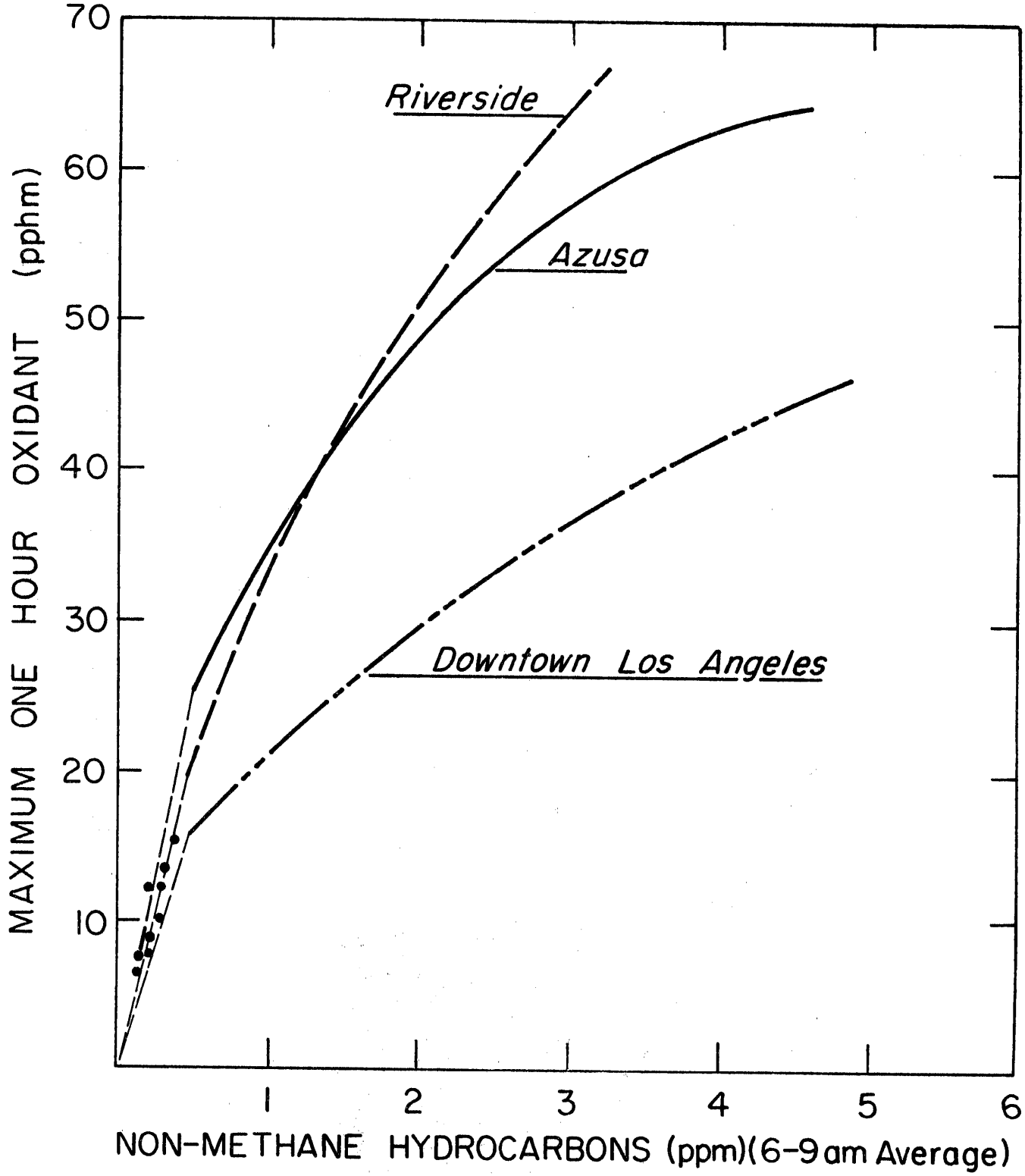


Figure 2. Upper limit one hour oxidant curves for selected South Coast Air Basin stations; 1968-1971 data.

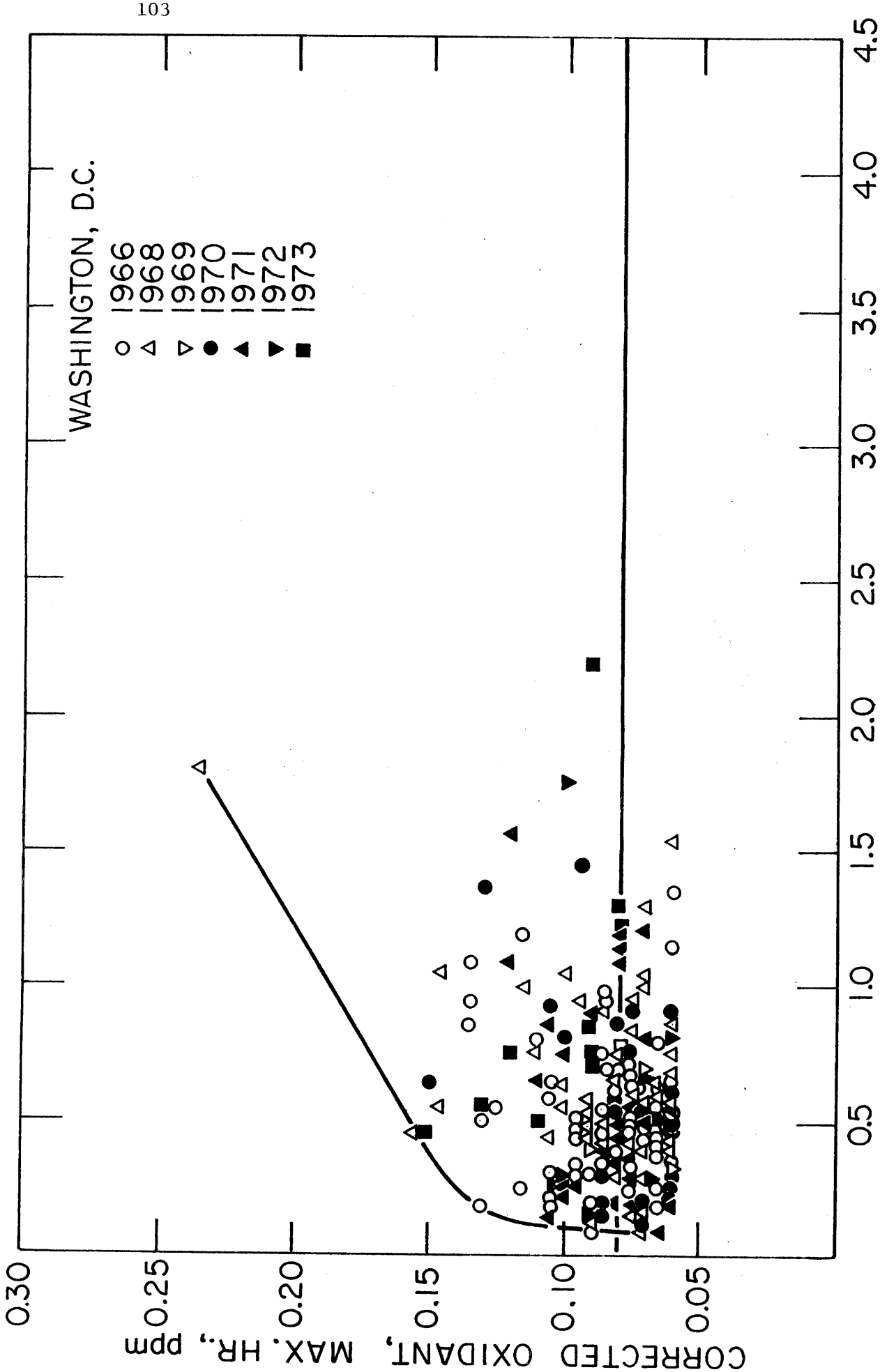

WASHINGTON, D.C.
1966
1968
1969
1970
1971
1972
1973
CORRECTED OXIDANT, MAX. HR., ppm
0.30
0.25
0.20
0.15
0.10
0.05
6-9 AM NON-METHANE HYDROCARBON, ppmC
0.5
1.0
1.5
2.0
2.5
3.0
3.5
4.0
4.5

Figure 3

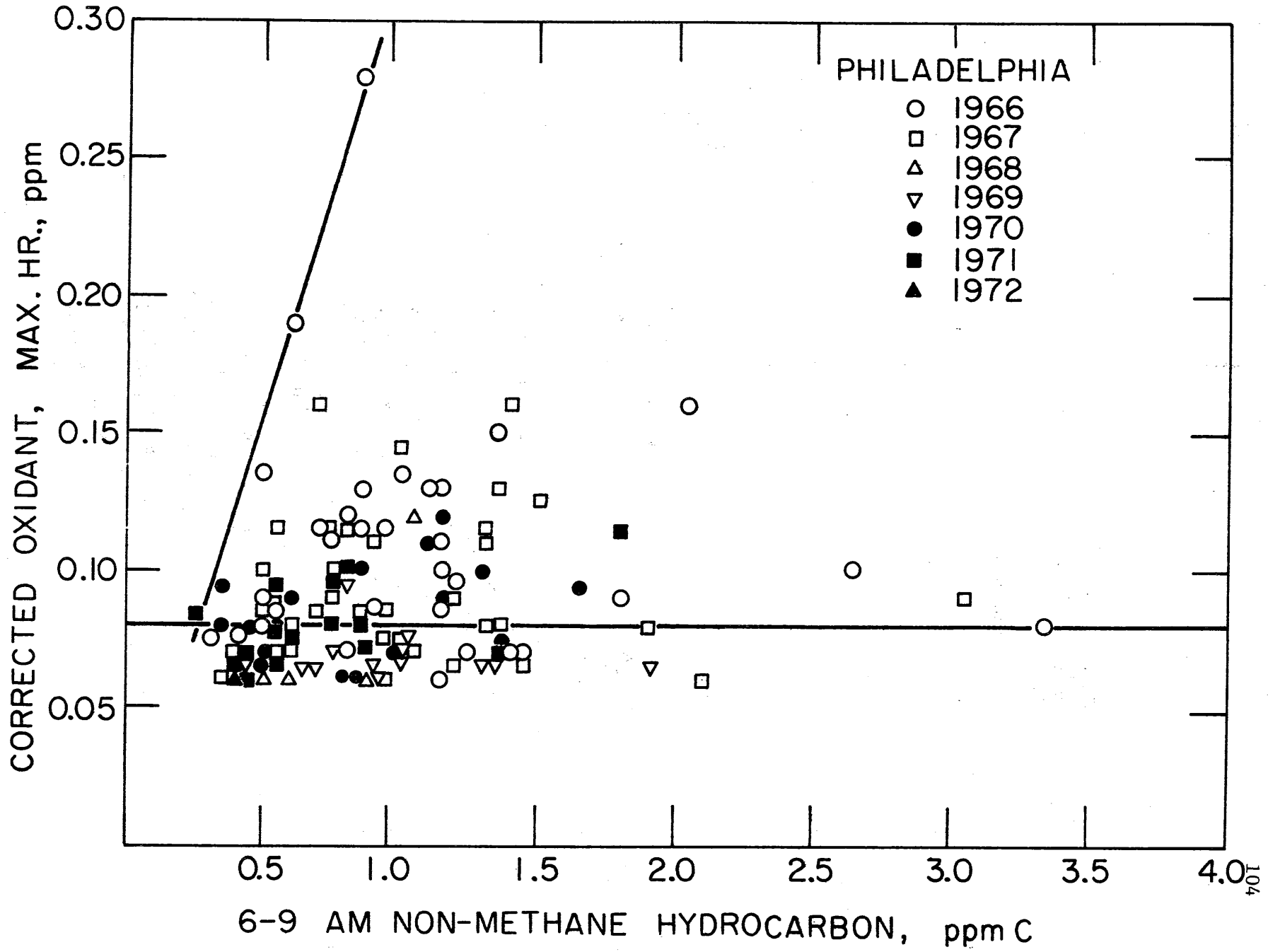

PHILADELPHIA
1966
1967
1968
1969
1970
1971
1972
CORRECTED OXIDANT, MAX. HR., ppm
0.30
0.25
0.20
0.15
0.10
0.05
0.5
1.0
1.5
2.0
2.5
3.0
3.5
4.0
6-9 AM NON-METHANE HYDROCARBON, ppm C
104

Figure 4

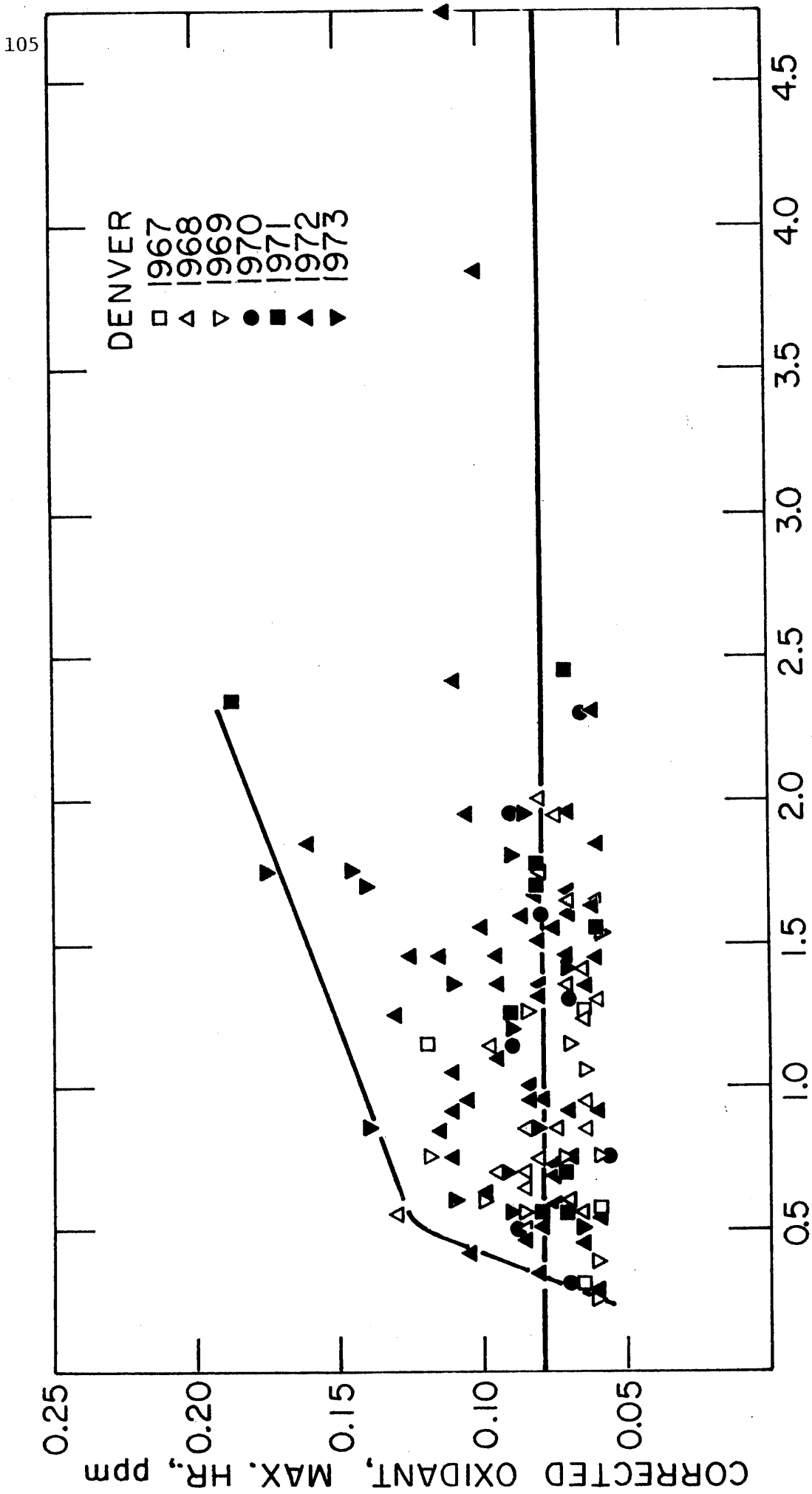

DENVER
1967
1968
1969
1970
1971
1972
1973
CORRECTED OXIDANT, MAX. HR., ppm
0.25
0.20
0.15
0.10
0.05
6-9 AM NON-METHANE HYDROCARBON, ppm C
0.5
1.0
1.5
2.0
2.5
3.0
3.5
4.0
4.5

Figure 5

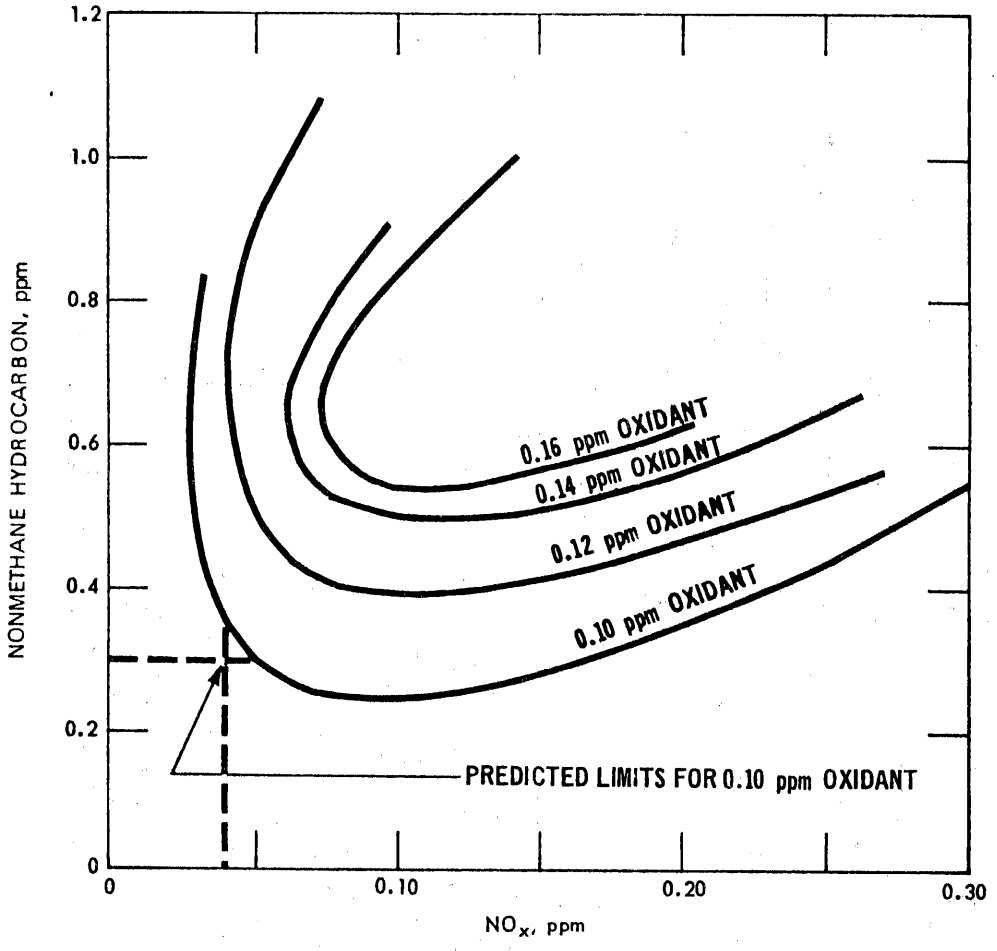


Figure 6. Approximate isopleths for selected upper-limit maximum daily 1-hour-average oxidant concentrations, as a function the 6-to 9-a.m. averages of nonmethane hydrocarbons and total nitrogen oxides in Philadelphia, Washington, D.C., and Denver, June through August, 1966 through 1968.

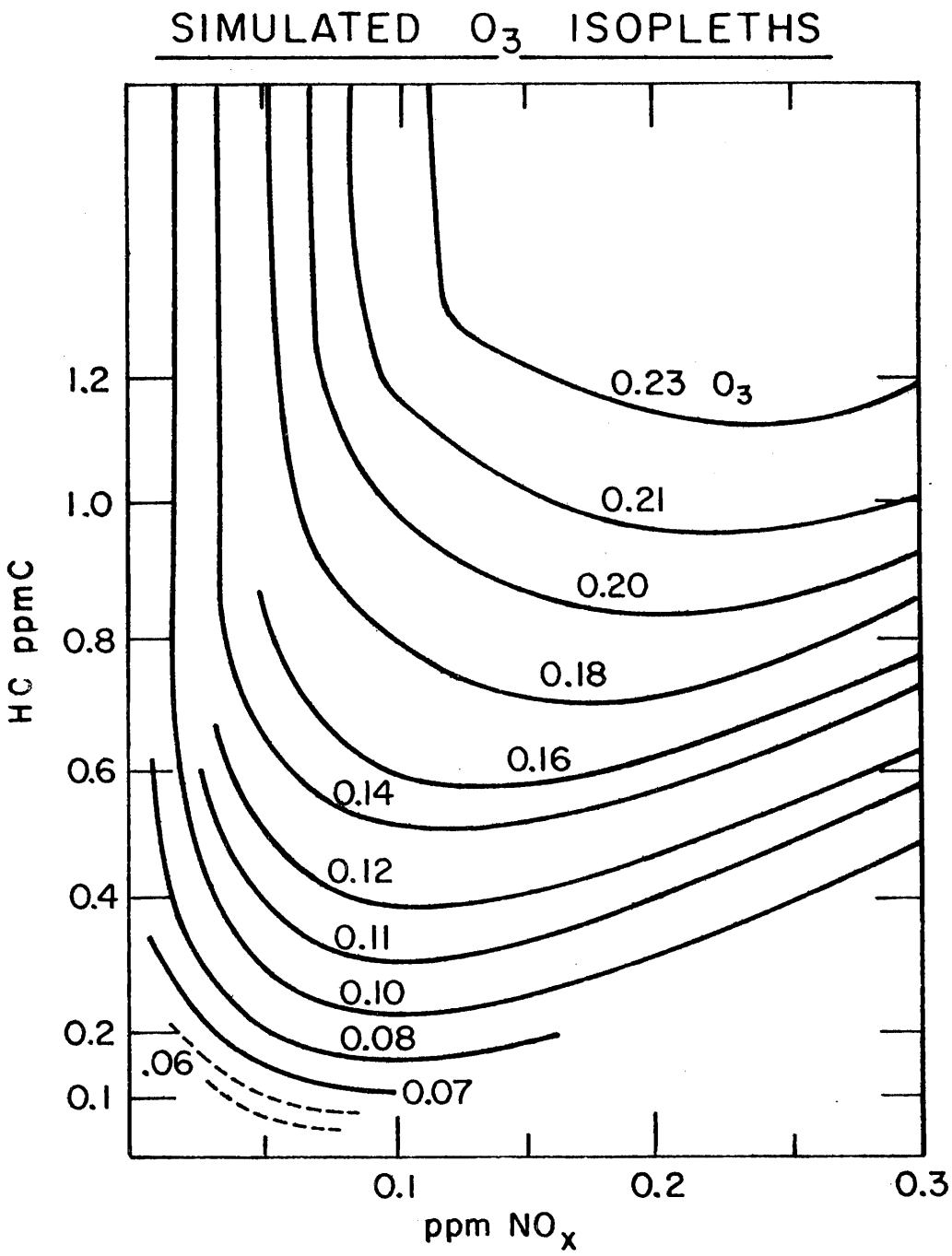

SIMULATED O_3 ISOPLETHS
0.23 O_3
0.21
0.20
0.18
0.16
0.14
0.12
0.11
0.10
0.08
.06
0.07
1.2
1.0
0.8
0.6
0.4
0.2
0.1
HC ppmC
0.1
0.2
0.3
ppm NO_x

Figure 7

THE VALIDITY OF THE STRATEGY OF LINEAR ROLLBACK OF HYDROCARBONS TO ACHIEVE OXIDANT AIR QUALITY STANDARDS

by

Robert E. Neligan
Director, Monitoring and Data Analysis Division
Office of Air Quality Planning & Standards
Environmental Protection Agency
Research Triangle Park, North Carolina

Good morning. The presentation I will make today is not entirely related to automobile emission controls. Rather it will address the technical basis for control strategies, automotive emission controls, pollutant transport models and control strategy formulation.

In Dr. Pitt's letter he had asked that we defer from going into all of the history of the particular subject that we were to address. However, I will briefly discuss a few of the pros and cons of linear rollback. The pros, it's simple, understandable, and it requires limited data input. The cons, its application is best for stable pollutants, and all variables such as source emission, meteorology, etc. are presumed to be consistent. However, linear rollback has never been scientifically validated, and it at best is a theoretical concept which we believe has some validity. I would have to categorize it as a first approximation. However, in most instances where an emission reduction percentage must be calculated there is no other alternative but to use linear rollback in many cases. In the past few years there have been some changes made in the linear rollback model. One of the major changes has been the disaggregation of sources in the model which has been described in the Morris/deNevers paper given at the last National Air Pollution Control Association Meeting. This modification was made to overcome one of the major criticisms of linear rollback, that all emission sources, either mobile or stationary had to be controlled to the same degree. Using the modified rollback technique one can disaggregate emissions and assign to each source category a specific emission reduction factor and growth rate. It is my opinion that the development of the modified linear rollback model improved this technique, however it does not overcome the fact that any rollback model has been validated under actual use. A further complication has arisen in the use of the rollback models when used for secondary pollutants such as oxidants, and that is the long distance transport of ozone and/or its precursors. When secondary pollutants and their precursors are transported over long distances, it becomes very difficult to select the receptor site where the maximum ozone concentration is likely to occur.

I would therefore like to discuss some of our recent findings concerning long distance transport of ozone and/or its precursors. We'll get away from Southern California for the next ten minutes or so and go to Ohio and Western Pennsylvania where the study was performed. May I have the first slide, please.

Going back to 1970, EPA conducted a pollution impact study of a power plant near McHenry, Maryland. The emissions from this electric power plant were affecting Christmas tree farms in that area and were believed to be causing pine needle injury and slow growth. While the aerometric measurements were mainly sulfur dioxide and particulate matter it was also decided to measure oxidants during the study. Surprisingly, rather high oxidant concentrations were measured. When I say high, relatively high for the eastern part of the U.S., .10 to .14 parts per million. The question naturally arose as to what was the source or sources of the oxidants as the study site was rather far removed from any major source of hydrocarbon emissions. First it was believed that ozone could be coming from the power transmission lines. A study was performed to see if they produced appreciable quantities of ozone. The measurements proved that the transmission lines were not the sought after source. The study ended, however the question as to reason for the high oxidant concentrations remained unanswered. EPA, therefore, went back the next summer to again investigate this rather intriguing question. The study took place during the summer months and it was found that during stagnating meteorological conditions, high concentrations of ozone (.10 to .15 parts per million) were again experienced. In the investigation of the wind trajectories it was determined that when winds were coming off the Eastern seaboard, or had passed over the Ohio and Indiana industrialized area, high ozone concentrations were experienced. In 1973 we iniated a study to look at a larger area that included McHenry, Maryland; Lewisburg, West Virginia; Coshockton, Ohio; and Kane, Pennsylvania. High ozone concentrations were again measured and it was found to be higher at these rural sites than the urban monitoring sites in the same region. Violations of the

oxidants standard were also found to be more frequent at the rural oxidants stations. In the 1974 study, six urban ozone sites were established at Cincinnati, Canton, Cleveland, Pittsburgh, Columbus, and Dayton. Rural monitoring sites were established at Wilmington, McConnellsville, and Wooster, Ohio; and Dubois, Penn. The site at McHenry was also continued for comparative purposes with past studies.

Second slide please.

This slide presents a summary of the ozone data collected at all sites for the month of July. It wasn't surprising that again high concentrations were found at the rural sites. The maximum concentration at Wilmington for example was .18 parts per million, at McConnelsville, .12 parts per million. The rural sites maximum concentrations were found to be relatively the same as the urban values which ranged from 0.12 to 0.18 parts per million. When the number of hours in which there was a violation of air quality standard for ozone were considered, it was found that the rural sites are having violations more frequently than the urban.

Slide No. 3, please.

I want to show you photographs of the rural monitoring sites as we are often asked, "Well, what do you consider to be rural?" This is the airport at Wooster in which there is less than 100 landings of aircraft per month or about 3 per day. It is approximately 60 miles east, southeast of Wilmington. As you can see, it looks like it is located in the center of a corn field and it was.

Slide No. 4, please.

This is the airport at McConnelsville, again a rural area, 50 to 60 miles from any major metropolitan area in Ohio, and with less than 100 aircraft landings per month.

Slide No. 5, please.

I would like to point out some of the diurnal variations and concentrations for nitrogen dioxide measured at the rural sites that were found.

I believe it is particularly notable that the nitrogen dioxide concentrations were never found to be very high. The maximum concentration was about .01 parts per million with an average of about .005 parts per million. The NO_2 instruments used in the study could not measure adequately in this range, as their detection limit was approximately .01 parts per million. In every instance when ozone above the 0.10 parts per million was measured, the associated NO_2 level was less than .01 parts per million and NO was essentially non-detectable.

Slide No. 6, please.

Methane on the other hand, usually was measured at the 1.5 to 1.7 parts per million range with total hydrocarbons values running in the range of 2.0 to 2.3 parts per million and remained fairly constant, over the study period.

Slide No. 7, please.

The slides that I have shown to you previously were data taken from ground monitoring stations. Aerial monitoring was also performed during this period of time in order to define the extent of high ozone concentrations. This slide depicts just one of a number of aerial flights that were made. The area investigated covered an area bounded by Wilmington, Ohio, in the west, Wooster, Ohio to the north, McHenry, Maryland, to the south and DuBois, Penn., in the east. The area covered by the flight was about 200 miles to the east and 100 miles to the north. During the flight ozone measurements were taken at 2 minute intervals. Hydrocarbons and carbon monoxide were measured by taking integrated bag samples at each numerical point shown on the chart for a total of 8 samples during the flight.

Slide No. 8, please.

The meteorological conditions during the flight are depicted on the next two slides. At 0700 the air was quite stable up to around 1,000 feet, with a subsidence inversion layer at about the 12,000 ft. level. A high pressure system was approaching from the west and

moving into the area at that time. As I recall, the wind speeds were fairly low and the high pressure system was moving northeastwardly at a rate of 8 to 10 knots per hour.

Slide No. 9, please.

At 1900 there was no evidence of a ground inversion and the top of the mixing layer was approximately 6000 ft. The altitude of the aircraft during the entire flight was 4000 ft. above mean sea level. It had therefore remained below the subsidence level and in the mixing layer during its entire flight.

Slide No. 10, please.

Ozone concentrations measured during the flight are shown on this slide. At Wilmington, concentrations were already at the air quality standard for photochemical oxidants, which is 0.08 parts per million or 160 $\mu g/m^3$. By the time the flight was over the McConnellsville area, concentrations had increased to approximately 250 $\mu g/m^3$ and varied between 160 and 390 $\mu g/m^3$ as the aircraft passed over Wooster and Dover. The lowest concentrations measured were at McHenry where levels of 140 $\mu g/m^3$ were measured. As the aircraft approached DuBois concentrations were at the 350 $\mu g/m^3$ level. During this period of time the winds were generally from a southeasterly direction. By early afternoon the aircraft was located about 25 miles northwest of Pittsburgh and the ozone concentrations had increased to roughly 400 $\mu g/m^3$. As the aircraft proceeded back to Wilmington, ozone concentrations decreased back to a value of about 160 $\mu g/m^3$.

Slide No. 11, please.

The next two slides portray a 48 hour history of the air parcels arriving at the Wilmington and DuBois monitoring stations in the form of wind trajectories. As shown in this slide the air parcel over these sites at 8 a.m. had two different origins. For DuBois, the air parcel had been located over northeastern Pennsylvania 48 hours previously, having moved in a clockwise direction. For Wilmington, the air parcel had also moved in a clockwise direction out of west-central Pennsylvania.

Slide No. 12, please.

By the evening of August 21 some twelve hours later, the wind trajectories indicate that the air mass over DuBois had passed over the New York, New Jersey and Southern Pennsylvania area during the previous 48 hours. The Wilmington trajectory indicates that the air mass had taken a more southerly route over Southern Maryland, Virginia, West Virginia and South Central Ohio. As you recall from a previous slide there was a significant difference between the ozone concentration found in the Wilmington Ohio area and those measured in the Pittsburgh-DuBois area.

Slide No. 13, please.

It is impractical to show the results of all the data obtained from the 8 hydrocarbon samples that were taken and analyzed as a result of this aircraft flight because of the large number of organic compounds that have been identified and their concentration determined in each sample. I have selected two, acetylene and carbon monoxide. It is most interesting to note that the concentration levels of these two compounds generally follow the oxidant concentration as shown in a previous slide. When one compares the data collected near Wilmington and McHenry, which were relatively clean areas, all pollutant ozone, acetylene, and carbon monoxide are at their lowest concentrations. When areas that exhibited high concentrations of ozone such as northwest of Pittsburgh, one finds that both acetylene and carbon monoxide have increased four or five fold over the Wilmington and McHenry areas.

Slide No. 14, please.

This slide is an example of the data collected from the gas chromatographic analysis of the integrated ambient air samples taken during each of the aircraft flights. Between 40 and 50 organic compounds may be identified using this technique. Evaluation and analysis of these data have only begun and efforts continue to identify the unknown compounds as they represent a significant portion of the total organic compounds measured.

Question: Can you say something about the relative molecular weight?

Answer: The unknowns for the most part are in the C8 to C10 range. The gas chromatographic technique used employed three separate G.C. columns. It was the third column, which was used for separation of the aromatic compounds as well as C8 and higher paroffinic compounds, that the unknown compounds appeared.

Question: Was this calibrated to methane?

Answer: That is correct.

I might digress a bit and state that I believe that one of the best quality control programs was performed this summer on all of the pollutant measurements taken. In general, it may be stated that the continuous measurements were within ±15% of the true value and in the GC analysis the maximum limits would run close to ±30%. I think this is very accurate at the part per million range.

Slide No. 15, please.

I have been presenting data based upon the aircraft flying at a constant altitude over wide areas. Vertical profiles were also flown particularly over the base station at Wilmington. This slide depicts the findings of three vertical flights taken on August 1. The first flight commenced just after dawn. At ground level ozone concentrations were approximately 60 $\mu g/m^3$. As the aircraft ascended through a rather strong temperature inversion layer the ozone concentration increased up to 160 to 170 $\mu g/m^3$. Between 2000 feet and 6000 feet the ozone concentration remained fairly constant and began to decrease above 6000 feet. The variance in these measurements may be interpreted as being due to destruction of ozone in the nocturnal radiation layer, preservation of ozone, with only slight destruction, in the previous day's mixing layer above the radiation inversion and lower concentration further aloft.

At mid-day, the ground inversion layer had broken. Vertical mixing plus the synthesis of ozone had resulted in increased ozone concentrations

to around 190 $\mu g/m^3$ at ground level going up to say about 210 $\mu g/m^3$ at 6000 feet. Above 6000 feet, there is again a decrease in concentrations. Again, I don't know whether this increase is significant with the knowledge that instrument accuracy is within ± 15% of the stated values. The late afternoon flight shows that the ozone concentration had continued to increase up to a level of 250 $\mu g/m^3$ from the ground up to 6000 feet at that altitude, decreasing values were again noted. The relatively high ozone values found late in the afternoon are typical of rural ozone patterns, with the daily maximum value usually occurring just before sunset. These vertical flights would seem to indicate that relatively high ozone concentrations can occur aloft during the night particularly when a noctural inversion isolates the air aloft from the ground. In turn, this ozone, produced the previous day, can fumigate the ground the next day as the noctural inversion layer is broken. Presumably organic compounds, which are precursors to ozone, are also present and would continue their photochemical reaction the next day.

Slide No. 16, please.

I would now like to suggest some 9 research endeavors as I believe they would provide some of the answers concerning the photochemical smog problem which in turn could lead to better control strategies.

Particularly, we need improved instrumentation for the measurement of low-level non-methane hydrocarbons. Gas chromatographic analysis is very expensive and not for all air pollution control agencies because of the costs of operation. To date, we have not found in our studies particularly good correlation between non-methane hydrocarbon analyzers and GC analysis. Oxides of nitrogen measurement appear to be accurate and reliable down to the .01 part per million level, however in our rural measurements, it was found that the NO_2 concentration rarely exceeded this level and was usually below .005 part per million. What is now required is an instrument that has precision and accuracy down to the part per billion level. Another requirement is a better understanding

of the nature and magnitude and temporal changes of hydrocarbons and oxides of nitrogen both from anthropogenic as well as natural sources. In addition it would be particularly valuable to know the temporal emission patterns of vehicles, so that hourly and daily changes can be estimated. I believe this is very critical to a better understanding of the synthesis of ozone. A third item would be an effort to tag specific hydrocarbons to specific stationary sources such as acetylene use as a specific indicator of internal combustion engines. The enhancement of the use of various freons as indicators of anthropogenic pollution is certainly a good start and should be expanded upon. There is little information on the qualification of organic material released by natural sources much less quantification. Much research work need to be performed in this field. The fourth item is a continuing need for irradiation chamber studies that Dr. Altshuller and Dr. Dimitriades spoke about yesterday. I believe that there is a need to continue the progress that is presently being made in irradiation chamber technology as it changes from a static to dynamic operation. Certainly a dynamic mode would be more representative of what is happening in the ambient atmosphere.

Slide No. 17, please.

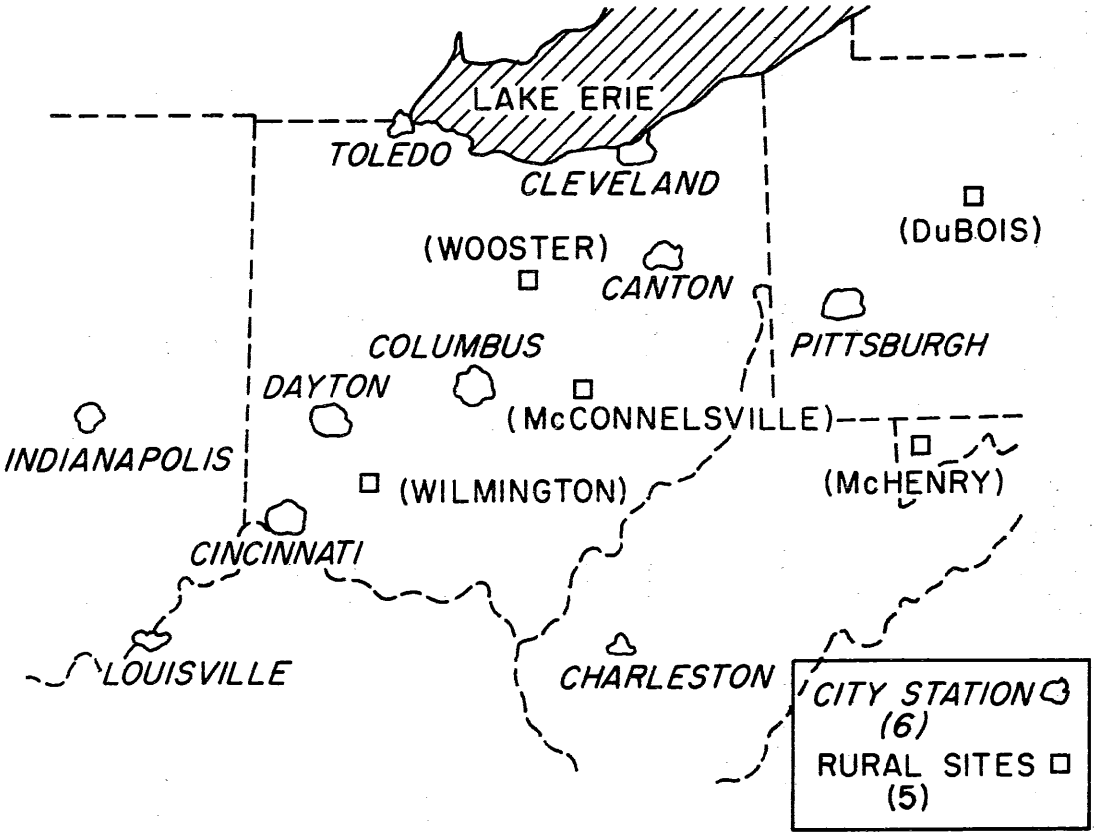
The fifth item I would like to address is the need for better instrument siting criteria. There is still little understanding of the extent of the area surrounding the monitor station that may be represented by measurements taken at that station. Such criteria should also be related to specific pollutants as well as their special and temporal distribution. The sixth item would be a continuation of studies to better define the mechanics associated with the long distance transport of ozone and or its precursors. Can the findings of past studies be related to other areas where the meteorological regimes are different? We need to find out!

Little is known about the fate or end products of the photochemical system. The large difference between urban and rural hydrocarbon/oxides of nitrogen ratios would suggest that the oxides of nitrogen may be removed from the photochemical system more rapidly than do the hydrocarbons. If so what is their end product or products?

Item 8 calls for further development of statistical models. Several models that have recently been developed, such as the Box-Jenkins time series, have shown much promise in relating pollution control efforts to resulting improvement in pollutant concentrations. The use of such models would be of great benefit to the control strategist as he could know that certain control efforts have resulted in a significant decrease in pollutant levels while others were only partially effective or not effective at all.

The final item would be the development and validation of regional photochemical models. By regional, I mean that the model could effectively estimate the pollution levels that would occur in an area of roughly 100,000 to 300,000 square miles. It would also determine what are the end products of the photochemical system and how they are removed from the system. Of course, I realize that all of the items mentioned are difficult undertakings, for if they were easy we would already have the answers. So with that I'm back to the point again of linear rollback model with all of its criticisms. It is a first approximation and is valid when used in that context. However, what are the alternatives to it today if we were to completely abolish its use?

Thank you.


LAKE ERIE
TOLEDO
CLEVELAND
(DuBOIS)
(WOOSTER)
CANTON
PITTSBURGH
COLUMBUS
DAYTON
(McCONNELSVILLE)
INDIANAPOLIS
(WILMINGTON)
(McHENRY)
CINCINNATI
LOUISVILLE
CHARLESTON
CITY STATION
(6)
RURAL SITES
(5)

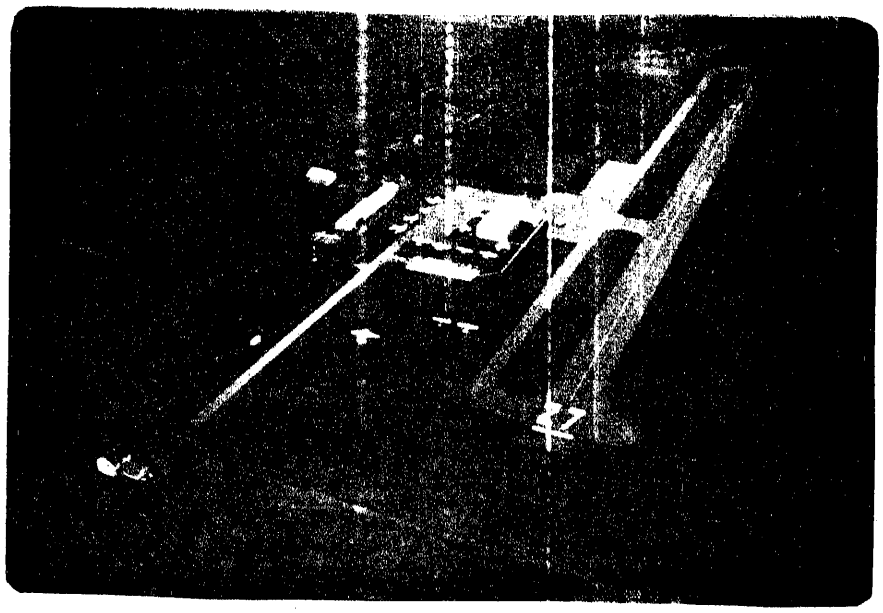
SLIDE No. 1

OZONE DATA
JULY 1974

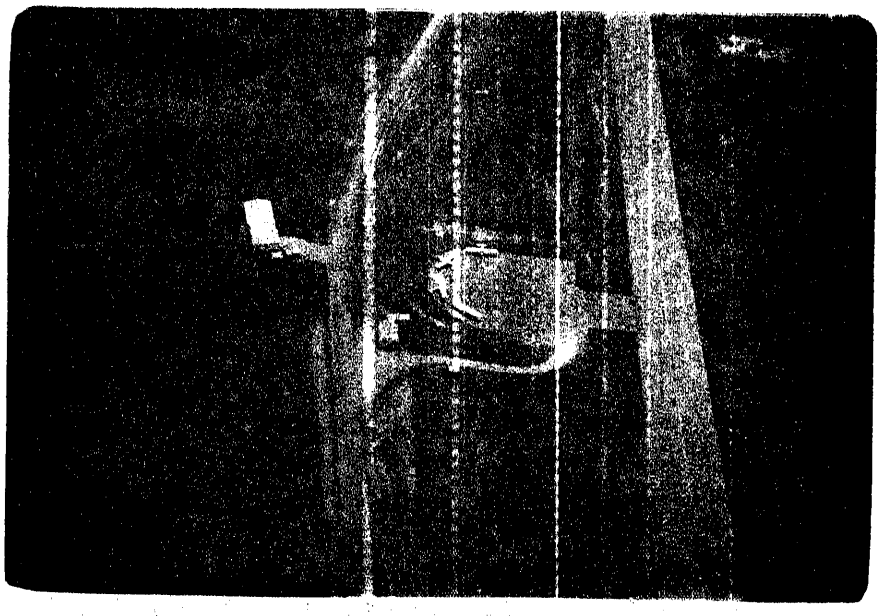
CITY	MAXIMUM CONCENTRATION ($\mu g/m^3$)	99TH PERCENTILE ($\mu g/m^3$)	DAYS EXCEEDING STANDARD (%)	HOURS ABOVE STANDARD (TOTAL)
		RURAL		
WILMINGTON, OH.	360	280	83	159 HOURS
McCONNELSVILLE, OH.	240	220	55	107 HOURS
WOOSTER, OH.	340	240	74	154 HOURS
McHENRY, MD.	340	300	63	160 HOURS
DuBOIS, PA.	400	360	86	235 HOURS
		URBAN		
CINCINNATI, OH.	360	260	28	28 HOURS
DAYTON, OH.	260	240	50	90 HOURS
COLUMBUS, OH.	300	250	58	101 HOURS
CANTON, OH.	260	220	55	115 HOURS
CLEVELAND, OH.	280	200	48	55 HOURS
PITTSBURGH, PA.	300	280	61	86 HOURS

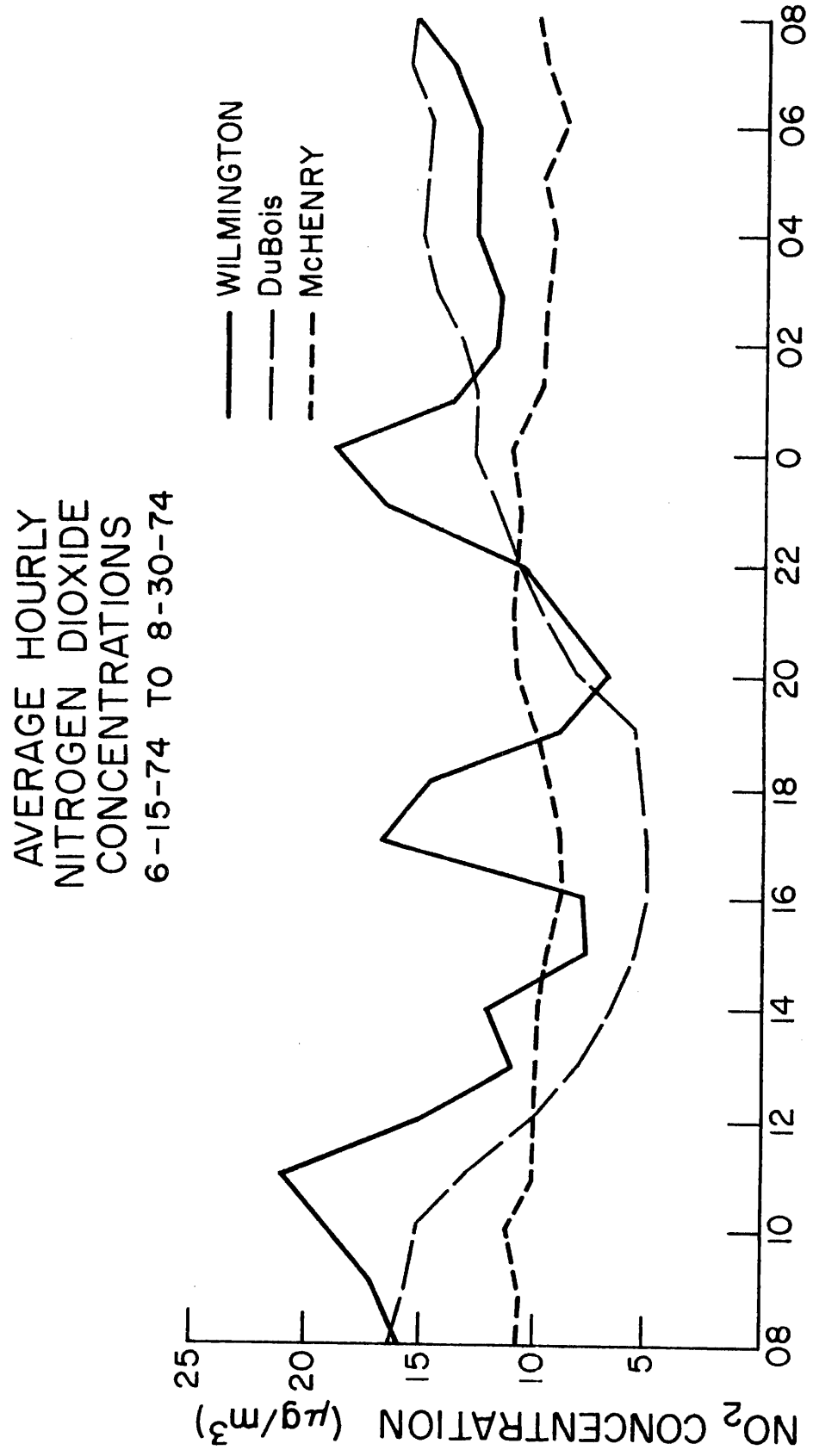
SLIDE No. 2

SLIDE No. 3



SLIDE No. 4

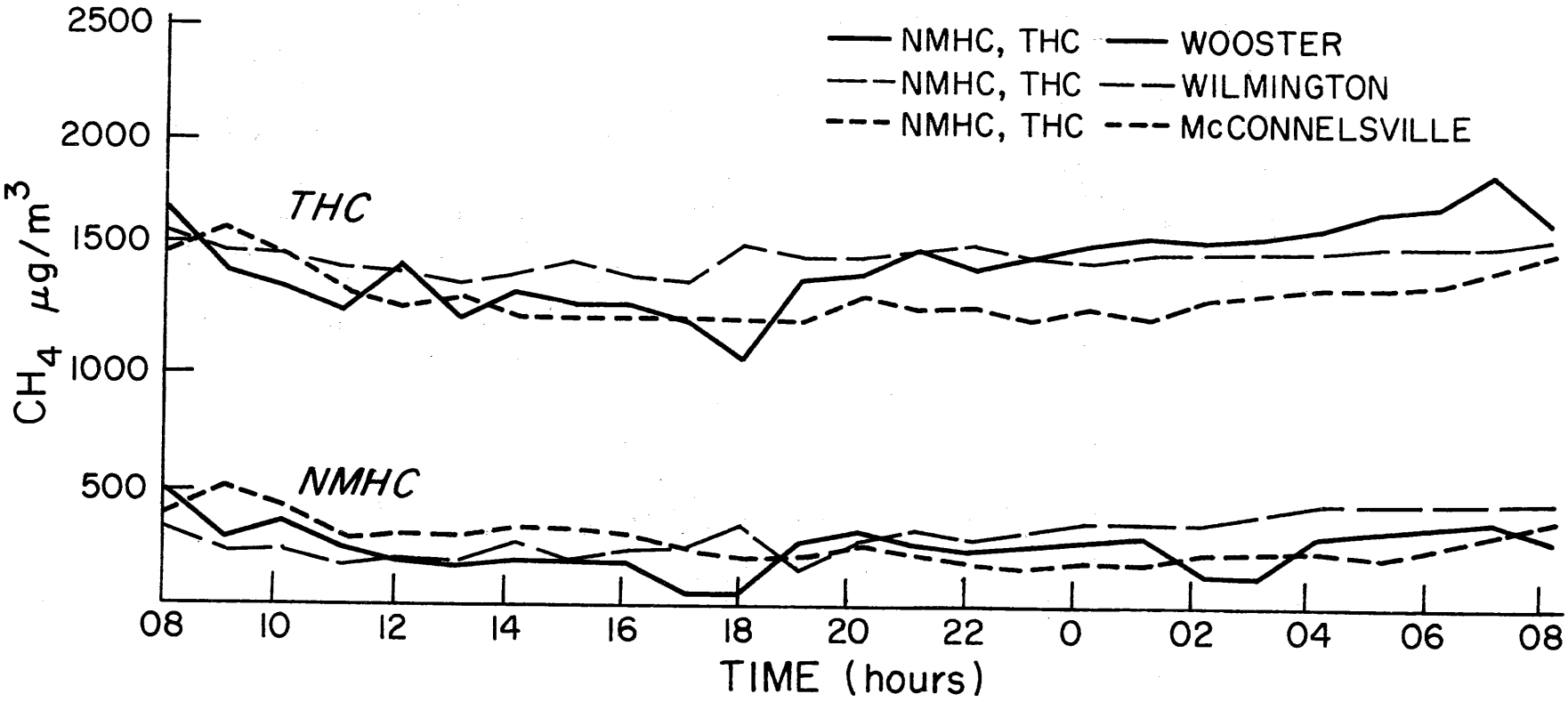



AVERAGE HOURLY
NITROGEN DIOXIDE
CONCENTRATIONS
6-15-74 TO 8-30-74
WILMINGTON
DuBois
McHENRY
NO2 CONCENTRATION (μg/m3)
25
20
15
10
5
08
10
12
14
16
18
20
22
0
02
04
06
08
TIME (hours)

SLIDE No. 5

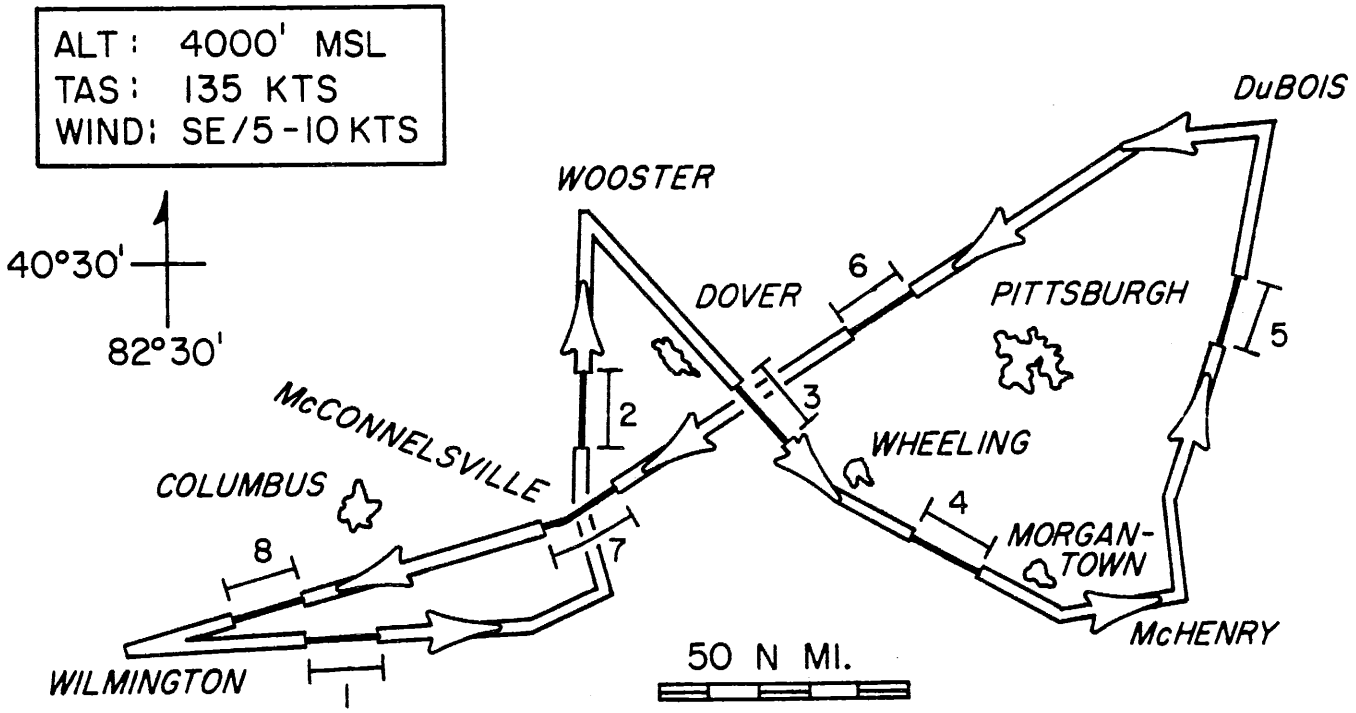
SLIDE No. 6

AVERAGE HOURLY TOTAL AND NON-METHANE HYDROCARBON CONCENTRATIONS JULY 1974

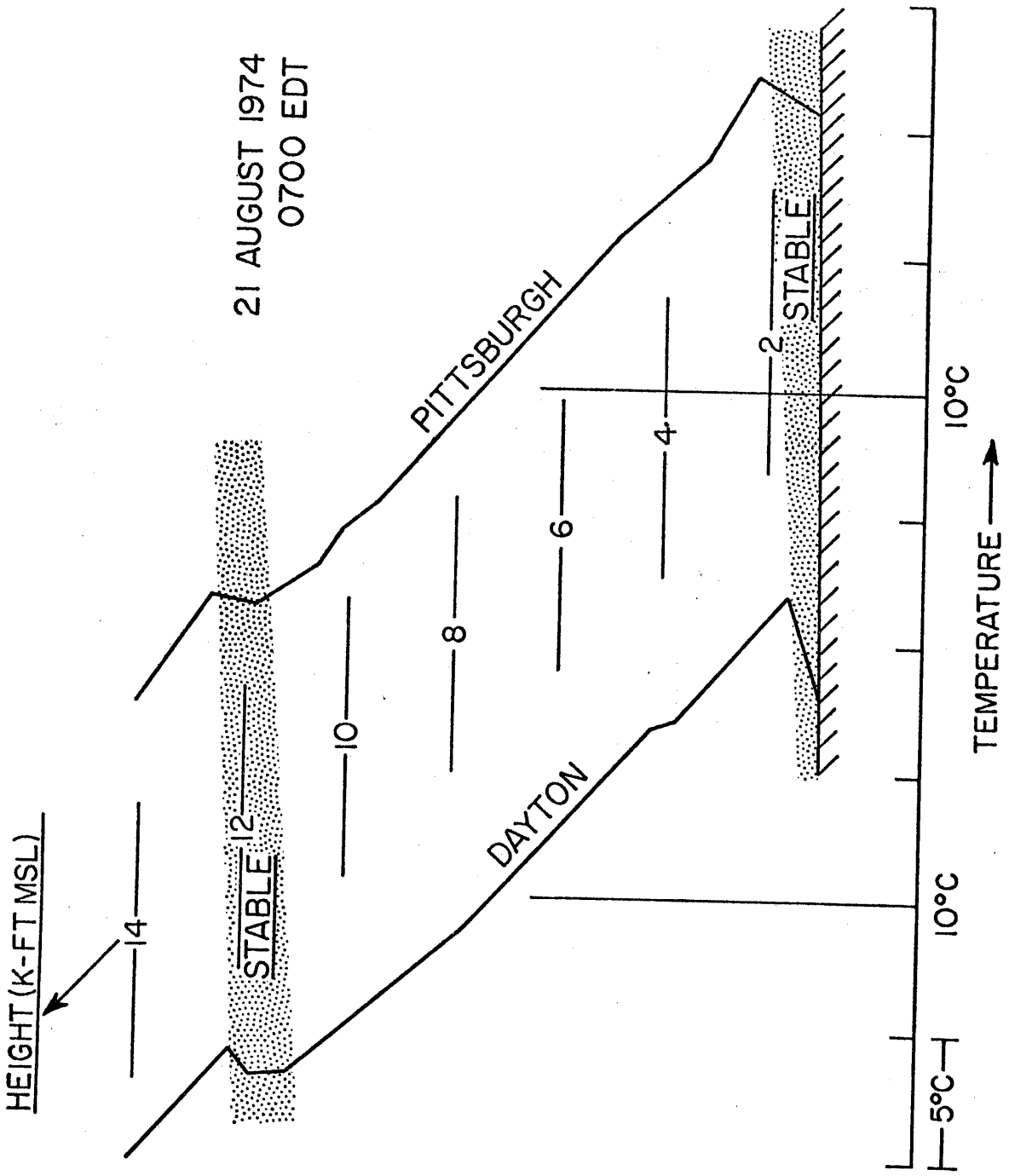


HYDROCARBON GRAB SAMPLES (6 MIN.)

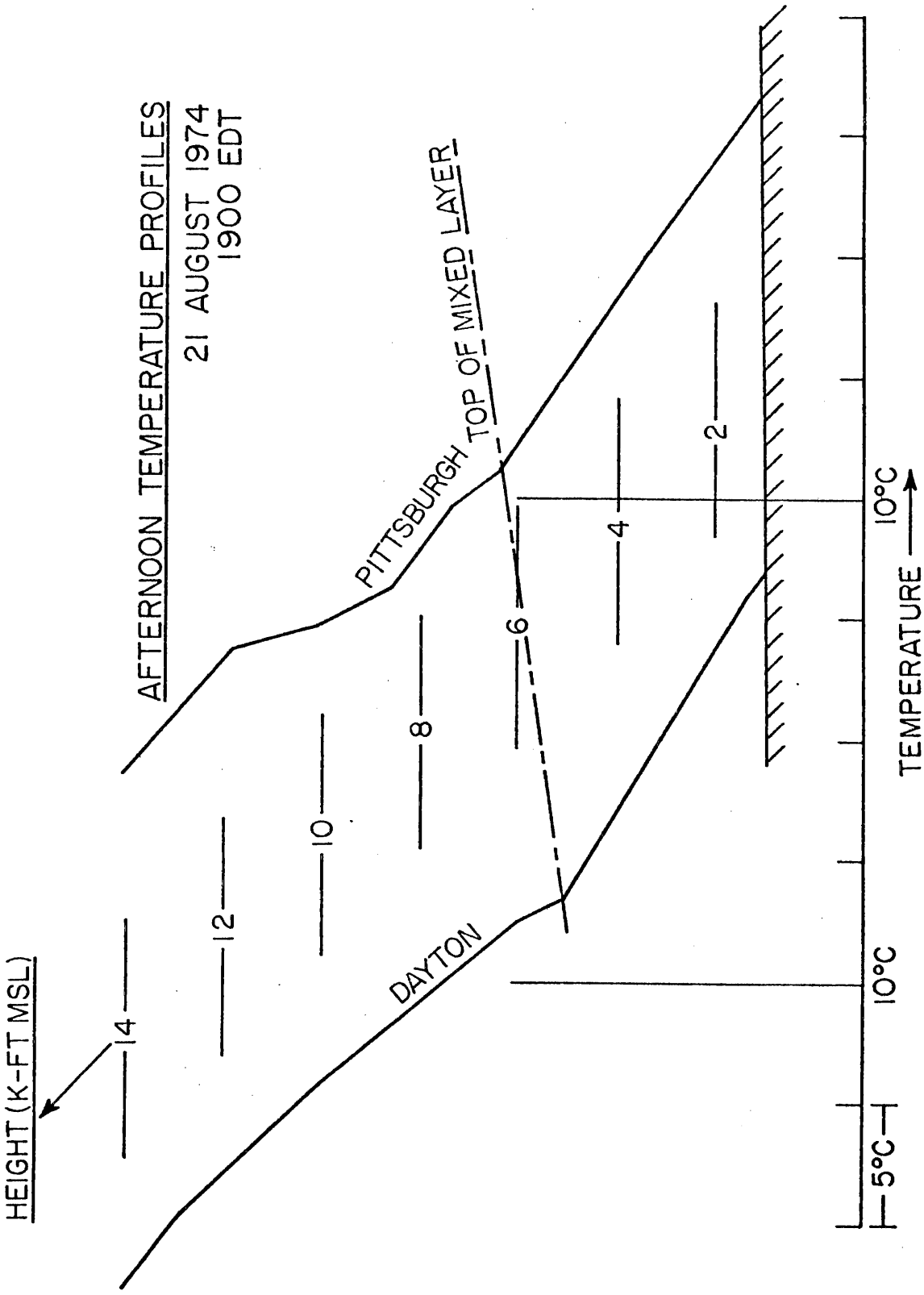
8-21-74



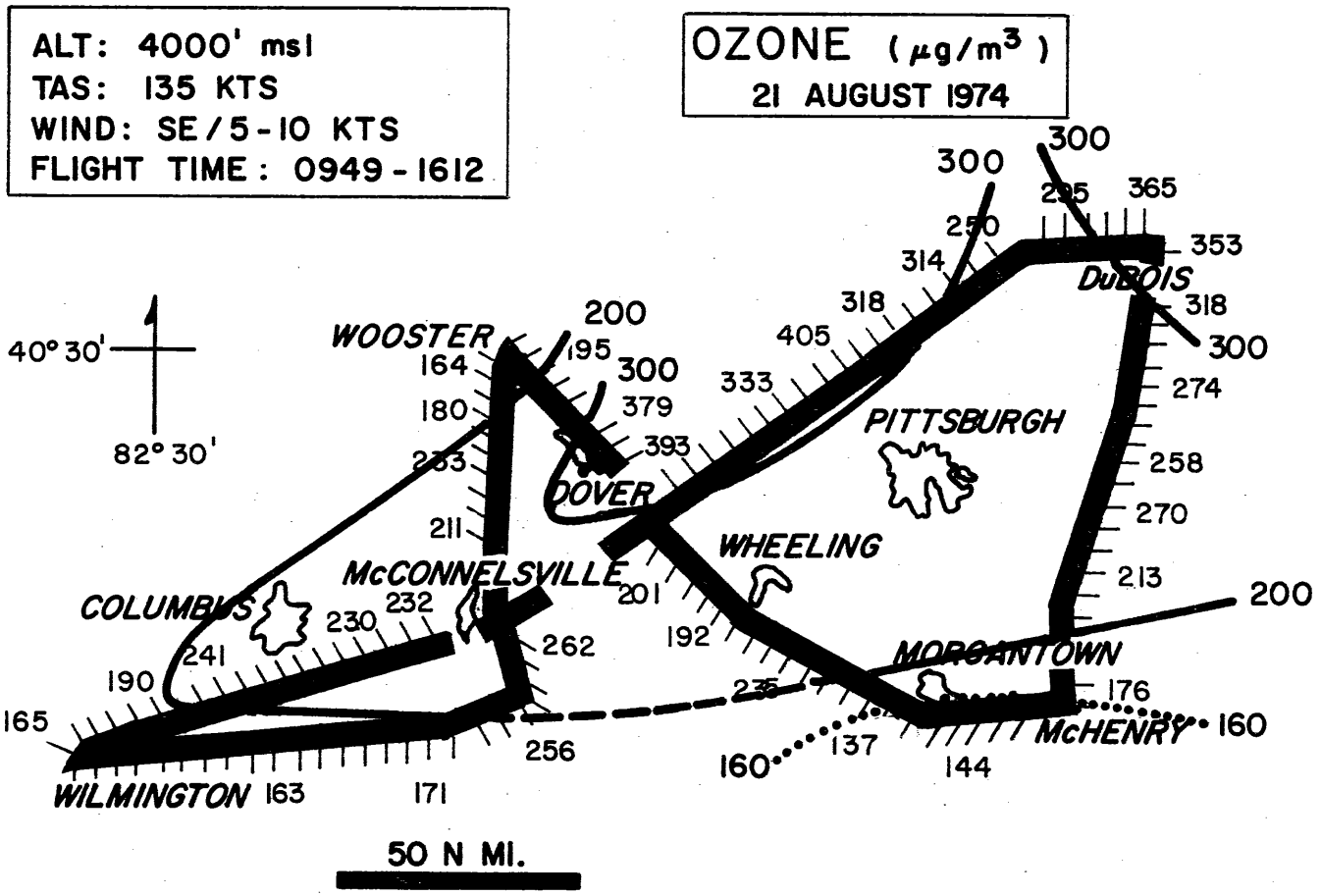
SLIDE No. 7


21 AUGUST 1974
0700 EDT
HEIGHT (K-FT MSL)
14
12
10
8
6
4
2
STABLE
STABLE
PITTSBURGH
DAYTON
10°C
10°C
5°C
TEMPERATURE

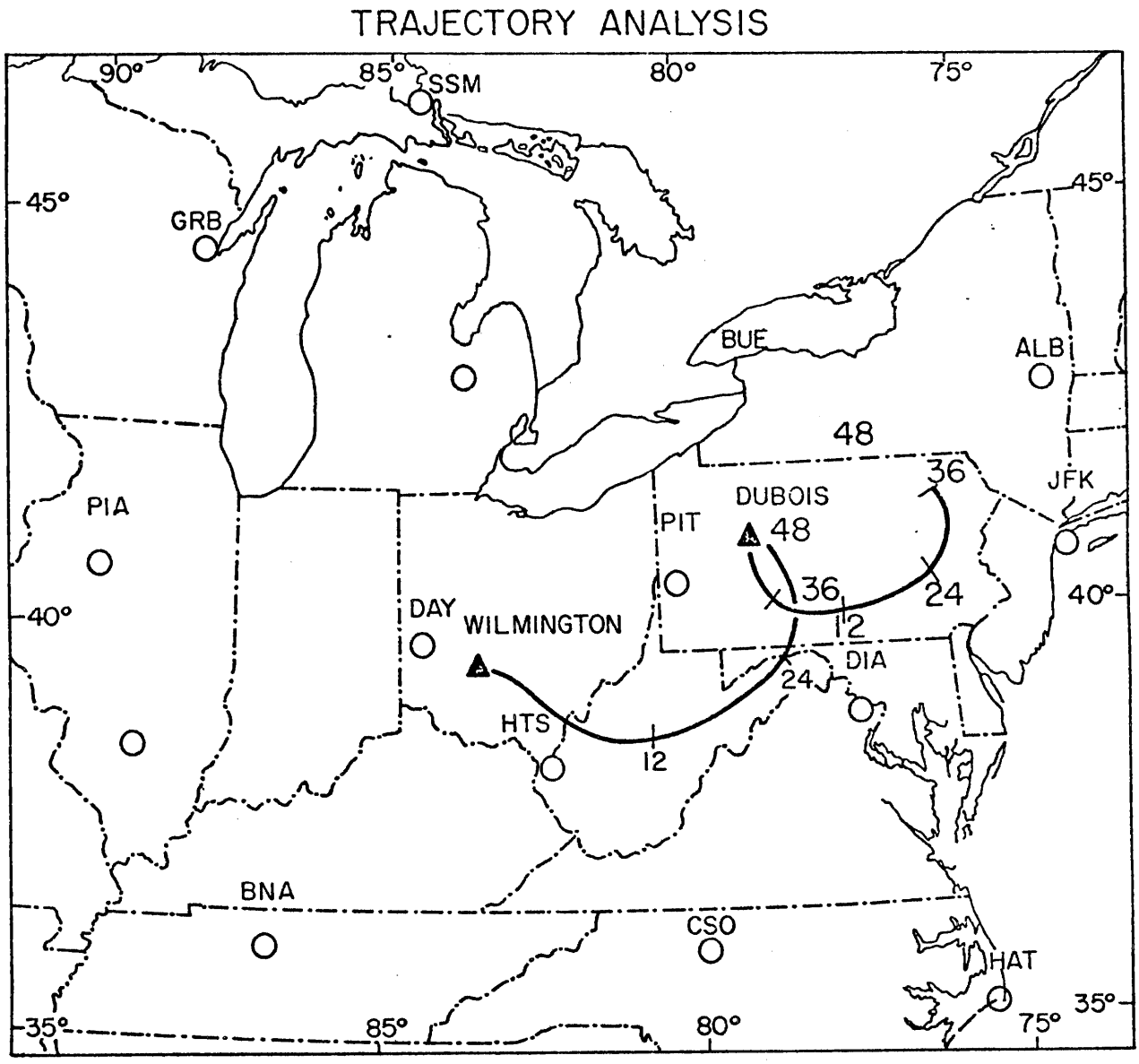
SLIDE No. 8


AFTERNOON TEMPERATURE PROFILES
21 AUGUST 1974
1900 EDT
PITTSBURGH
TOP OF MIXED LAYER
DAYTON
HEIGHT (K-FT MSL)
14
12
10
8
6
4
2
5°C
10°C
10°C
TEMPERATURE

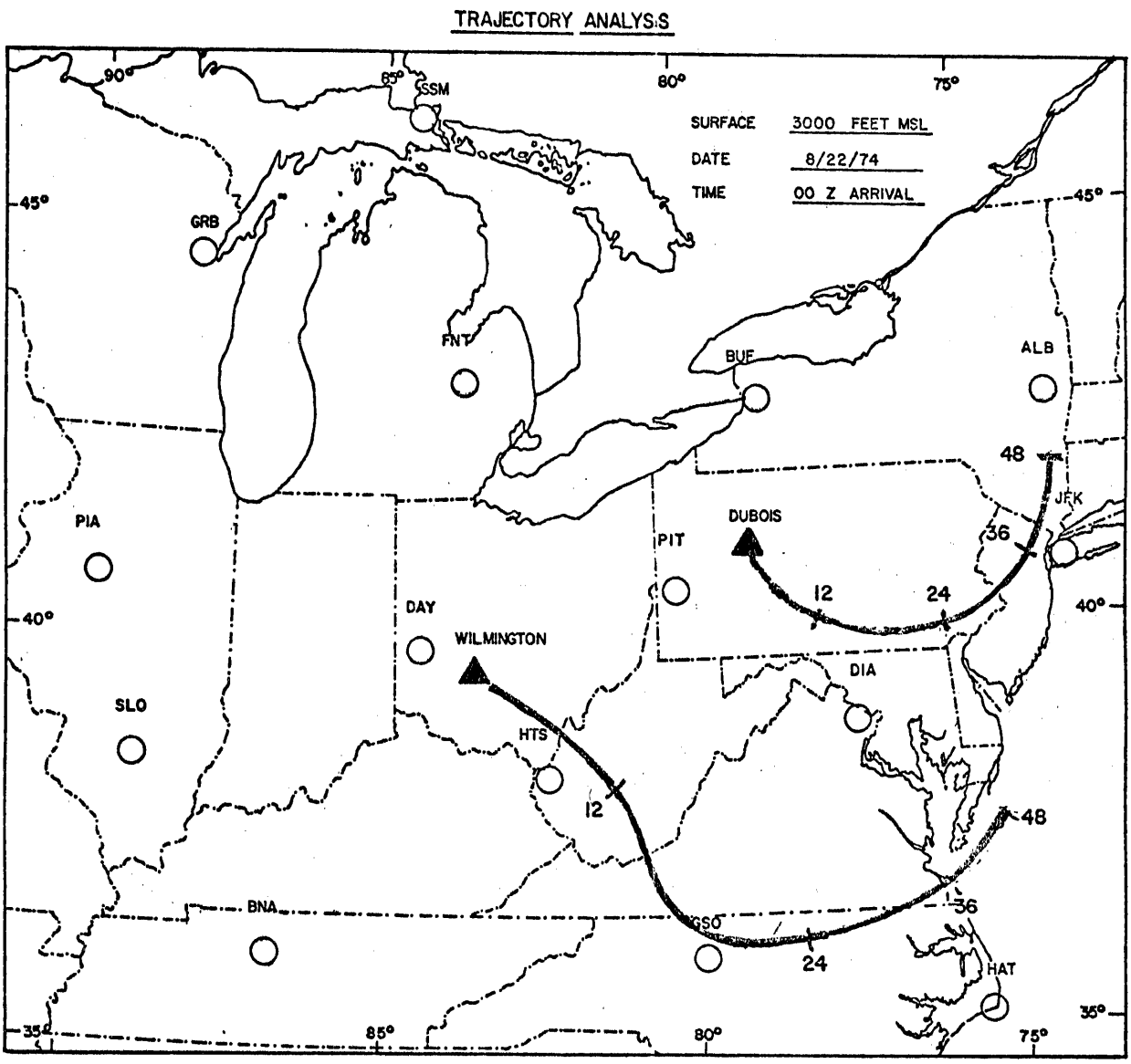
SLIDE No. 9


ALT: 4000' msl
TAS: 135 KTS
WIND: SE/5-10 KTS
FLIGHT TIME: 0949-1612
OZONE (μg/m³)
21 AUGUST 1974
40° 30'
82° 30'
WOOSTER
DuBOIS
PITTSBURGH
DOVER
WHEELING
McCONNELSVILLE
COLUMBUS
MORGANTOWN
McHENRY
WILMINGTON
300
365
353
318
274
258
270
213
200
176
160
144
137
235
192
201
262
256
232
230
241
190
165
163
171
160
314
318
405
333
379
393
211
180
164
195
200
50 N MI.

SLIDE No. 10

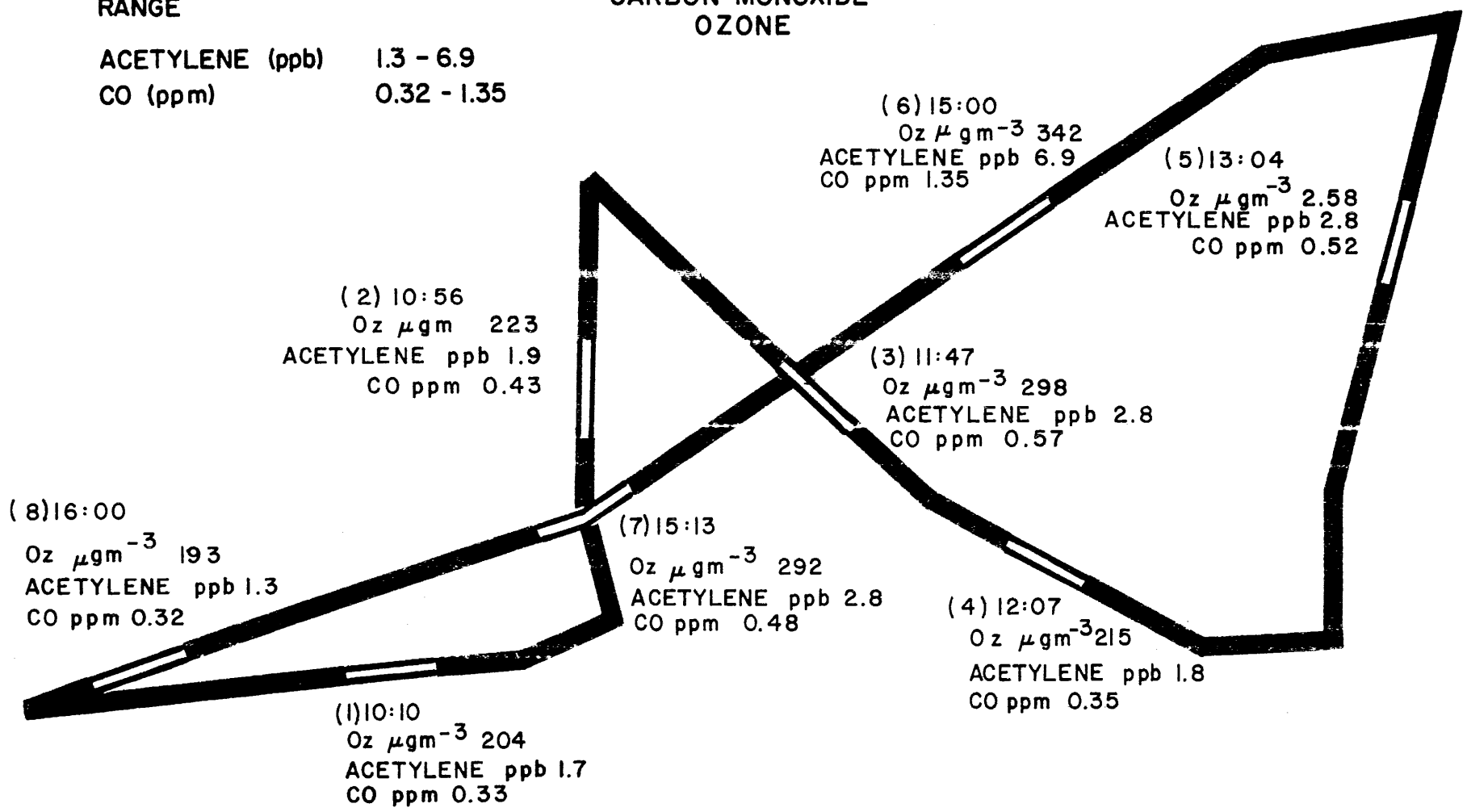

TRAJECTORY ANALYSIS
90°
85°
80°
75°
45°
40°
35°
SSM
GRB
BUE
ALB
JFK
PIA
PIT
DUBOIS
48
36
24
12
DAY
WILMINGTON
DIA
HTS
BNA
CSO
HAT

SLIDE No. 11


TRAJECTORY ANALYSIS
SURFACE 3000 FEET MSL
DATE 8/22/74
TIME 00 Z ARRIVAL
90°
85°
80°
75°
45°
40°
35°
SSM
GRB
FNT
BUF
ALB
JFK
PIA
PIT
DUBOIS
DAY
WILMINGTON
DIA
SLO
HTS
BNA
GSO
HAT
12
24
36
48

SLIDE No. 12

HYDROCARBONS
AIRCRAFT FLIGHT
AUG. 21, 1974
ACETYLENE
CARBON MONOXIDE
OZONE



SLIDE No. 13

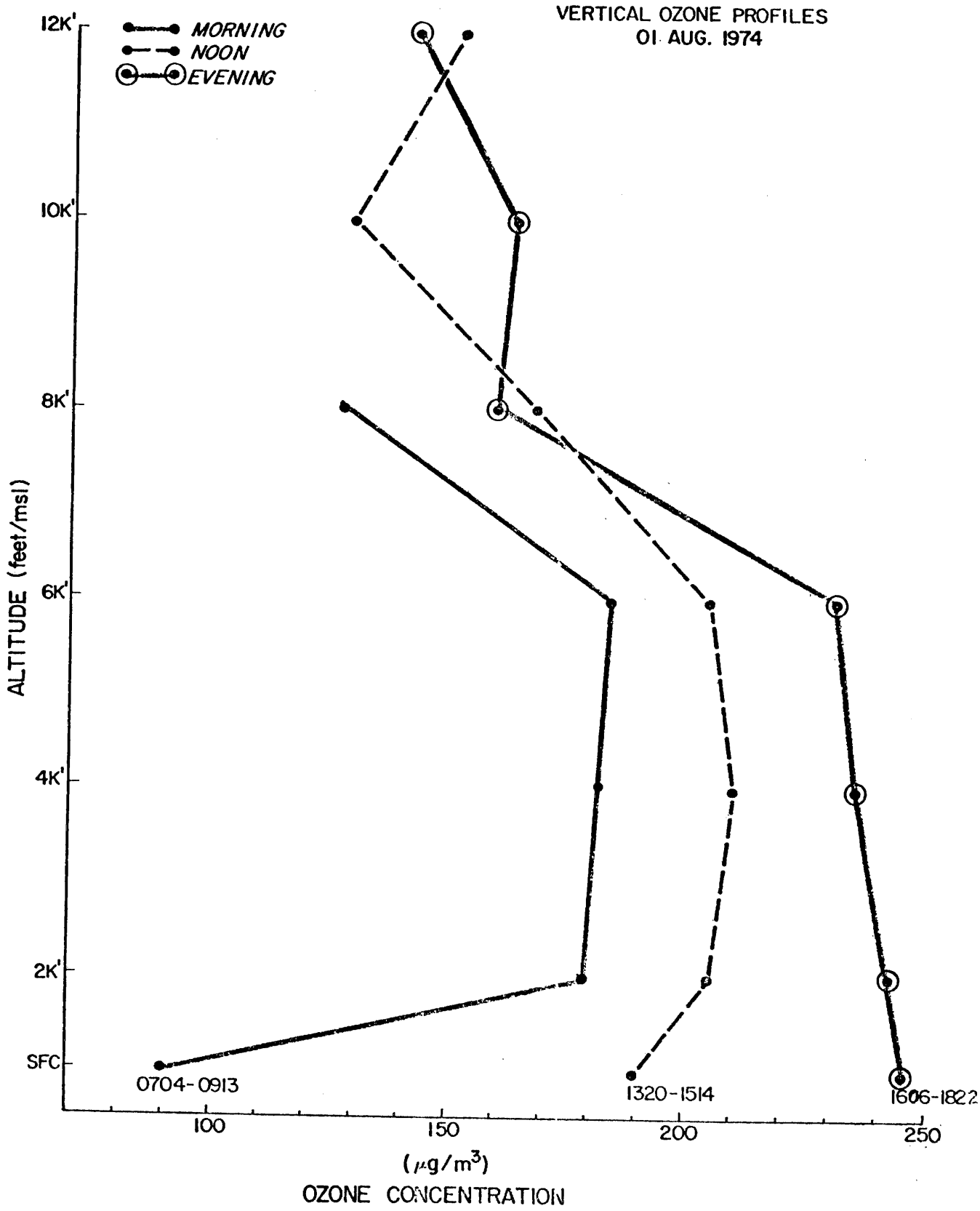
HYDROCARBON ANALYSIS (PPBC)
EPA PERKIN ELMER MODEL 900

SAMPLE NO. 6--AUGUST 21, 1974 AIRCRAFT FLIGHT

SATURATED COMPOUNDS		OLEFINS		AROMATICS		CARBONYL COMPOUNDS		UNKNOWNS	
ETHANE	8.5	ETHYLENE	3.7	TOLUENE	12.5	ACETALDEHYDE	6.5	UNKNOWN (C8)	9.4
PROPANE	6.0	ACETYLENE	6.9	ETHYLBENZENE	2.5	ACETONE	20.5	UNKNOWN	7.3
N-BUTANE	14.8	PROPYLENE	1.9	P-XYLENE	1.5			UNKNOWN	0.5
ISOPENTANE	14.4	ISOBUTYLENE	1.4	M-XYLENE	2.5			UNKNOWN	1.3
N-PENTANE	6.4	TRANS-2-BUTENE	0.8	O-XYLENE	12.6			UNKNOWN	28.0
CYCLOPENTANE	1.7	CIS-2-BUTENE	0.3	N-PROPYLBENZENE	0.6			UNKNOWN	5.2
2-METHYLPENTANE	5.1	1-PENTENE	0.4	M & P-ETHYLTOLUENE	2.3			UNKNOWN	57.3
3-METHYLPENTANE	4.8	B-PINENE	1.4	1,3,5 TRIMETHYLBENZENE	1.1				
N-HEXANE	3.0			O-ETHYLTOLUENE	0.4				
CYCLOHEXANE	0.4			1.2.3 TRIMETHYLBENZENE	1.0				
2-METHYLHEXANE	1.3								
2,3-DIMETHYLPENTANE	0.8								
3-METHYLHEXANE	1.5								
2,2,4-TRIMETHYLPENTANE	1.2								
N-HEPTANE	1.7								
METHYLCYCLOHEXANE	1.7								
NONANE	1.8								
N-DECANE	1.5								

METHANE	1.70 PPM (BECKMAN 6800)
TOTAL HYDROCARBONS	1.93 PPM (TOTALED FROM ABOVE DATA)
CARBON MONOXIDE	1.35 PPM (BECKMAN 6800)

SLIDE No. 14


VERTICAL OZONE PROFILES
01 AUG. 1974
MORNING
NOON
EVENING
12K'
10K'
8K'
6K'
4K'
2K'
SFC
ALTITUDE (feet/msl)
0704-0913
1320-1514
1606-1822
100
150
200
250
(μg/m³)
OZONE CONCENTRATION

SLIDE No. 15

SLIDE No. 16

(1) IMPROVED INSTRUMENTATION FOR THE MEASUREMENT OF LOW LEVELS OF NON-METHANE HYDROCARBONS AND OXIDES OF NITROGEN.

(2) A BETTER UNDERSTANDING OF THE NATURE, MAGNITUDE, AND TEMPORAL CHANGES OF HYDROCARBON AND OXIDE OF NITROGEN EMISSIONS, BOTH MAN-MADE AND NATURAL. THIS WOULD INCLUDE METHODS FOR DETERMINING AREA-WIDE TRAFFIC DENSITIES AND TEMPORAL-SPATIAL CHANGES IN VEHICLE MILES TRAVELED.

(3) THE DETERMINATION OF HYDROCARBON COMPOUNDS THAT ARE SPECIFIC TO EMISSION SOURCES WHICH IN TURN COULD BE USED TO DETERMINE THE RELATIVE MAGNITUDE OF SUCH EMISSIONS IN THE ATMOSPHERE. THIS WOULD REQUIRE THE DEVELOPMENT OF DETAILED GAS CHROMOTOGRAPH TECHNIQUES AND THEIR SUBSEQUENT USE IN BOTH SOURCE AND AMBIENT ANALYSES.

(4) ADDITIONAL LABORATORY AND RADIATION CHAMBER STUDIES THAT CAN REFLECT ATMOSPHERIC CONDITIONS AS CLOSELY AS POSSIBLE.

SLIDE No. 17

(5) THE DEVELOPMENT OF BETTER CRITERIA FOR THE SITING OF AIR MONITORING INSTRUMENTS.

(6) ADDITIONAL MONITORING STUDIES, BOTH BY AIRCRAFT AND GROUND STATIONS TO BETTER DEFINE THE LONG DISTANCE TRANSPORT OF OZONE AND/OR ITS PRECURSORS.

(7) INVESTIGATIONS TO DETERMINE THE NATURAL SINKS FOR NITROGEN DIOXIDE AND OXIDANTS.

(8) STATISTICAL MODELS SHOULD BE FURTHER EXPLORED AND DEVELOPED. SUCH MODELS UTILIZING THE TIME SERIES APPROACH, SUITABLY MODIFIED TO ACCOUNT FOR FUTURE EMISSION PATTERNS COULD SIGNIFICANTLY ADVANCE THE STATE-OF-THE-ART IN CONTROL STRATEGY DEVELOPMENT.

(9) AS NEW DATA INPUTS ARE ACQUIRED AND DOCUMENTED, THE FURTHER REFINEMENT AND DEVELOPMENT OF REGIONAL DIFFUSION MODELS.